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Diarrhea of the Newborn 969
Stewart H. Clifford

An Unusual Case of Acute Infective Polyneuritis with Visceral Lesions 976
Frederic Parker, Jr., and Raymond D. Adams

Emphysematous Gastritis 983
Claude E. Welch and Chester M. Jones

What Industry Thinks About the Massachusetts Medical Society's Program of Medical Care for the People of Massachusetts 986
Jarvis Hunt

Massachusetts Department of Public Health 1004

Medical Progress: The Nasal Carrier of Beta-Hemolytic Streptococci 988
Henry M. Lemon

Case Records of the Massachusetts General Hospital 995

Editorials:

January 1, 1948 1002

Pandora's Box 1002

The Massachusetts Medical Society and the Boston Medical Library 1003

Subscription Rate Increases 1004

Notices 1004

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DIARRHEA OF THE NEWBORN*

Its Causes and Prevention

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BOSTON

A REVOLUTIONARY change in social custom concerning childbirth has occurred during the past decade. Local evidence of this change is afforded by the statistics from the Boston Lying-in Hospital. This hospital delivers about 20 per cent of the infants born in Boston each year. The total number of babies delivered in this institution in 1935 and in 1945 was about the same: 4530 and 4685, respectively. In 1935 there were 1015 home deliveries in the outpatient department — in 1945 there were but forty-seven home deliveries, and in 1946 there were none.

The virtual disappearance of home births was not reflected in a corresponding increase in the deliveries on the wards of the hospital. In 1935 there were 2762 ward births, and in 1945 there were 2236.

What, then, happened to the missing 1500 patients? The hospital statistics furnish the answer to this question; these patients demanded private physicians and private ward care, and they were delivered on the private side of the hospital. Richardson House, originally designed to accommodate 900 patients, saw the birth of 753 infants in 1935 and 2401 in 1945.

There can be but one answer to this phenomenon: the public is convinced that the safest place for a mother to have her baby is a maternity hospital. So far as the mother is concerned statistics have proved the public to be right, for the maternal mortality is reaching an all-time low; it remains to be proved whether the hospital or the home is the safest place for a newborn infant. Evidence submitted from New York City demonstrates that there is at present a persistently upward trend in the death rates from diarrheal disorders in the neonatal group of infants.[1] This increase is due in great measure to the prevalence of epidemic diarrhea of the newborn.[1]

CAUSES OF EPIDEMIC DIARRHEA OF THE NEWBORN

It is significant that the literature on epidemic diarrhea of the newborn has appeared only during the past fifteen years. The appearance of this syndrome as a public-health problem coincides with the shifting of the scene of childbirth from the home to the hospital.

Countless factors have contributed to making the newborn infant in a lying-in institution extremely vulnerable to all types of infection. The combination of an increased birth rate and increased hospital births has led to the mass overcrowding of babies in nurseries. Shortage of personnel in the face of increased demands has resulted in a breakdown in nursery and formula-room technics.

Out of his long experience as a hospital consultant Pollock[2] observes the tendency of architects and consultants to fail to provide adequate space for nurseries. He found that "all too frequently nurseries are assigned to what might be designated as left-over space and their size is determined by the number of bassinettes that can be crowded in without any regard to spacing or the aisles between them."

A review of the literature clearly indicates that the syndrome described as epidemic diarrhea of the newborn is not a pathologic entity but a miscellaneous group of cases of various etiologies, known and unknown, bound together by a common symptom — diarrhea.

The reported epidemics can be divided into three groups. In the first, evidence of a bacterial origin is present, although the organisms discovered frequently belong to groups not ordinarily considered pathogenic; the infecting agent apparently enters the nursery via an adult carrier and reaches the infant by the fecal-oral route through a break in the nursery or formula-room technic. The second group

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includes the epidemics in which a virus etiology has been proved or strongly suspected with the backing of certain presumptive evidence. The large and controversial third group comprises outbreaks in which etiologic studies on the stools have failed to demonstrate an inciting agent.

Epidemics of Bacterial Origin

A significant investigation into the cause of recurrent outbreaks of epidemic diarrhea of the newborn in a Kansas Hospital has been reported by Ensign and Hunter,[3] whose approach was based on a knowledge of the method of spread of the infection rather than on a bacteriologic search for specific organisms. From June to October, 1945, six outbreaks of diarrhea had occurred in the hospital nursery; during the same period more than 400 cases of diarrhea in adults had occurred in the community. Prior to each of five hospital outbreaks, one mother or nurse on the obstetric floor had developed the disease. In the sixth outbreak the mother had had the disease shortly before entering the hospital. In every case in which the mother had the disease her infant developed it. The nurseries were crowded and there was a shortage of personnel. The authors reasoned as follows:

> The outbreaks could be easily explained on the basis of secondary infection from infected mothers or nurses if we keep in mind a well-known fact in the epidemiology of gastrointestinal disease, namely, that apparently healthy persons who have ingested organisms are carriers of those organisms until they are eliminated from the alimentary canal. These organisms may be eliminated in one or two days. Nevertheless, until they are eliminated, persons harboring them are temporary carriers capable of passing the infection on to others. The only condition which could result in a large group of temporary carriers is a recent ingestion of contaminated food or water.

Their investigations proved that the milk supply of the entire community was grossly contaminated with *Bacillus pyocyaneus* (*Pseudomonas aeruginosa*) at the dairy and that the milk was then not sufficiently pasteurized. Bacterial counts on the milk disclosed over 3,000,000 organisms per cubic centimeter. *Ps. aeruginosa* was isolated from all infants' stools cultured, from autopsy material, from nurses' stools and from the milk supply.

McClure[4] reports four different hospital epidemics of diarrhea of the newborn in the Province of Ontario. In Hospital "A" two outbreaks occurred. In the first of 62 infants, 26 became ill, and 3 died. Hemolytic *Escherichia coli* was found in the stool cultures of 14 of 16 infants and also in the nursery supervisor's stool culture. In the next outbreak, which involved 31 infants with 7 deaths, eight stool cultures were made, and seven showed hemolytic *Esch. coli*. Stool cultures of 16 normal infants were negative. Cultures in 4 of 17 nurses showed hemolytic *Esch. coli*. The milk formula showed a high bacterial count, with nonhemolytic *Esch. coli*. Cultures from the top of the common bathing table showed hemolytic *Esch. coli*. Six of the hemolytic *Esch. coli* strains from the infants, bathing table and a night nurse were identical by sugar reactions. Another strain from an infant and one from a nurse were identical.

In Hospital "B" the epidemic was dated from the time a mother and her infant developed diarrhea. Seven infants became ill, and 1 died. Subsequently, stool cultures on 12 healthy babies in the nursery revealed hemolytic *Esch. coli* in 7. In Hospital "C" 12 infants developed diarrhea, and an abundant growth of hemolytic *Esch. coli* was obtained from evaporated-milk formula and nipple jars. In Hospital "D" a severe epidemic occurred; 18 infants had the disease, and 12 died. Hemolytic *Esch. coli* were obtained from the stools of 8 of 11 sick infants and from 2 of 18 healthy infants. In their investigation the authors found the nursing technic in all these hospitals very faulty — the diapers were rinsed in the same nursery sink in which the formula bottles were subsequently warmed. Milk was stored on the wards in quart stock bottles from which the individual bottles were filled at feeding time.

Baker[5] reported three epidemics of diarrhea of the newborn occurring between 1935 and 1938 in one New York Hospital. The first outbreak involved 59 infants in the obstetric nurseries. Various organisms were grown on stool culture but not consistently enough to be thought significant. These organisms included *Esch. coli mutabile*, hemolytic *Staphylococcus aureus*, *Staph. albus*, *Bacillus subtilis*, *Esch. coli*, alpha-hemolytic streptococcus, gamma-hemolytic streptococcus and *Aerobacter aerogenes*. The second outbreak involved 13 premature infants. Cultures of the stools of 9 patients revealed *Esch. coli mutabile* or hemolytic *Esch. coli* organisms, which some workers have regarded as pathogenic. The author quotes Dulaney, who found *Esch. coli mutabile* in the stools of 67 per cent of patients in an epidemic of diarrhea of the newborn. In the third outbreak involving 13 infants, no suspicious stool organisms were isolated.

McKinlay[6] described an epidemic involving 6 infants with 2 deaths apparently caused by an organism of the Salmonella group. She found a nurse as the probable carrier of the infection. Bloxsom[7] reported an epidemic of infectious enteritis in Houston, Texas, with a mortality of 50 per cent. Stool cultures in 18 cases revealed organisms of the Salmonella group in 5. Abramson et al.[8] discussed an epidemic of diarrhea in the newborn due to Salmonella infection. Cummings[9] observed a nursery outbreak in Michigan caused by *Salmonella Panama*, with 12 deaths. Diarrhea had been rampant six months prior to the epidemic studied and had occurred sporadically in the interval. *S. Panama* was recovered from the stools of a graduate nurse, a nurse's aide and a maid.

Jampolis and his colleagues[10] have reported an epidemic due to *Klebsiella pneumoniae* (*Bacillus*

mucosus capsulatus). This infection was traced to 3 nursery maids.

Schwentker[11] described an epidemic of diarrhea from Baltimore in which there were 21 deaths. *Bacillus dispar* was recovered from the stools of 36 infants.

Wheeler and Foley[12] isolated Lancefield Group D streptococci in an outbreak in Boston, with epidemiologic and clinical studies that suggested respiratory or air-borne transmission. The authors make the important observation that Group D streptococci may be of pathogenic significance in infants. They point out that beta characteristics are not specific for this group of streptococci. Alpha, beta, and gamma strains were encountered in all serologic types of Group D organisms — "a fact which invalidates many earlier and more extensive attempts to determine the incidence of the various serologic groups of streptococci in normal infants."

The preceding review of recorded outbreaks of epidemic diarrhea demonstrates several points: the epidemics may be caused by a diversity of organisms; the newborn infant appears to be very susceptible to infection with organisms not ordinarily considered pathogenic; any infant fed milk or fluid from a bottle is peculiarly vulnerable to infection because the same person who feeds him and handles the nipples also handles the diapers and the infant's excreta, and it is well known that efficient scrubbing is required to free the hands and fingernails of bacteria of fecal origin; and many of the epidemics can be traced to carriers in the nursery personnel, infection by this fecal-oral route constituting ample evidence of faulty nursing technic.

Epidemics of Virus Etiology

A virus etiology for certain outbreaks of diarrhea in the newborn has been suspected from the first. However, the evidence for a virus infection has been chiefly negative, many epidemics having received most careful epidemiologic and bacteriologic investigations without the discovery of a specific etiologic organism. The infants in these outbreaks frequently exhibit a normal or lowered white-cell count and fail to respond to chemotherapy.

Lyon and Folsom[13] described three epidemics in 1926, 1934 and 1938 that suggest the virus of influenza as a possible etiologic agent, since at the time of each epidemic clinical influenza infection was present in the community.

In 1943 Campbell[14] reported from Melbourne outbreaks occurring in three hospitals at the same time that epidemic cases of so-called "gastric flu" were common in the city. In 1944 a severe epidemic of diarrhea of unknown etiology in the newborn was observed in Philadelphia by High, Anderson and Nelson,[15] occurring at the same time as an epidemic of diarrhea, nausea and vomiting of unknown cause in adults.

The first positive evidence that a filterable virus was a cause of certain epidemics of diarrhea of the newborn was submitted by Light and Hodes.[16] Two epidemics were studied in 1941, and stool filtrates tested in a variety of small animals, with negative results. In 1942 two epidemics in Baltimore and two in Washington were studied. In each of the four epidemics pooled stools, Seitz-filtered in three and unfiltered in one, were given nasally to young calves. In two to five days all the animals developed a bloody mucoid diarrhea that lasted an average of three weeks. Successive calf passages were easily made with each of the four strains. Cross-immunity studies indicated that the four strains represented a single agent.

After recovery the calves had developed immunity and large doses of active material failed to cause disease. Serums obtained from 6 infants after convalescence conferred complete or partial protection against the nasal injection of active material. The virus was not inactivated until the material had been boiled for ten minutes or exposed to a temperature of 80°C. (170°F.) for an hour.

Cummings[9] is convinced that he was able to recover, in an epidemic of diarrhea of the newborn, a filterable agent of the type described by Light and Hodes, although he was unable to maintain the potency of this agent beyond the fifth calf transfer. This epidemic involved 22 babies. Nose, throat and stool specimens were obtained free from bacterial pathogens. Pooled material was tested for viruses in mice, monkeys, guinea pigs, chicks, cotton rats, chick embryos, hamsters, newborn pigs and newborn calves. All the animals or mediums reacted negatively except the calves.

In October 1943 Buddingh and Dodd[17] observed one of a pair of twins to have stomatitis and diarrhea. Within the next two weeks and in spite of strict isolation precautions 16 of 30 infants in the nursery developed diarrhea, with a mild stomatitis in 10. In contrast with other epidemics of diarrhea of the newborn this outbreak was relatively mild, with no fatal cases. With an inoculum of stools obtained in this epidemic the authors were able to produce lesions on the scarified cornea of rabbits. The agent was readily filterable through Berkefeld V. candles. Strains of the agent were maintained for as many as forty-five serial passages. Immunity developed in the rabbit eye in a period of three weeks. Serum obtained from 12 patients during the acute disease produced positive results when mixed with the virus; similar mixtures using serum obtained during convalescence caused no lesions. Attempts to propagate the virus in other animals or in the developing chick embryo were unsuccessful. The filterable agent has been isolated by Buddingh[18] from stools obtained in epidemics in Nashville, Memphis, Cincinnati and Boston. Cross-immunity has been demonstrated between all strains regardless of whether the death rate in an epidemic was 0 or over 50 per cent. It has not been possible

to determine whether the viruses isolated by Light and Buddingh are the same. It seems probable that a virus is the usual etiologic agent in epidemic diarrhea of the newborn, but, as in encephalitis, different viruses may be concerned in different epidemics.[11]

Cummings[9] and the group studying diarrhea and enteritis in Michigan encountered an epidemic or diarrhea in the premature section of a children's hospital. Because newborn calves were not available at the time, the material obtained was tested on smaller animals with negative results and on the rabbit's cornea after the method of Buddingh and Dodd. The Seitz filtrates of pools of infants' stools gave positive eye reactions. However, subsequent experience with serial passages of corneal material led the group to conclude that the reaction was erratic if not nonspecific. They tested a large number of rabbit corneas with trauma, trauma and alundum, stools and mouth washings from normal infants and various bacteria and found it possible with all these specimens or technics to produce what they considered to be typical positive eye reactions as described by Buddingh and Dodd.

Cummings's conclusion that Buddingh's rabbit-eye scarification test is not specific for the virus of epidemic diarrhea is a great disappointment to all workers in the field. This simple method of proving a virus etiology in an epidemic under investigation had been expected to clarify much of the present confusion surrounding this syndrome. At present the test must be shelved until other investigators prove its exact status.

The lack of a relatively simple technic to identify the virus of this disease is the greatest handicap to further progress. Both Light and Hodes and Cummings agree that the virus can be grown and studied in newborn calves. This method is of limited usefulness in actual practice. Light and Hodes have reported but one experiment based on four outbreaks, whereas Cummings, with his elaborate study group financed jointly by the Kellogg Foundation and the Michigan Department of Health Laboratory in a four-year study of many epidemics of diarrhea, has only reported the use of calves in testing the material from one epidemic.

Since we cannot at the moment accept the epidemics reported by Buddingh and Dodd as having a proved virus etiology, the only positive evidence is found in the four epidemics reported by Light and Hodes and the one reported by Cummings.

Epidemics Not Associated with the Usual Bacterial Pathogens

The epidemics of diarrhea in the newborn in which no virus etiology has been demonstrated and in which none of the usual bacterial pathogens have been found constitute by far the largest group reported in the public press and in the medical literature and by public-health officials studying the problem.

An epidemic involving 32 infants with a high mortality was described in 1936 by Barenberg, Levy and Grand[19] in which no specific etiology could be found but in which the authors suspected that either a virus or certain toxins of the colon bacillus were responsible. Rice et al.[20] analyzed eleven outbreaks of highly fatal diarrhea in New York City between 1934 and 1936 in which no inciting agent could be discovered. In a subsequent paper in 1939 Frant and Abramson[21] confirm the negative pathological and bacteriologic findings and point out that diarrhea of the newborn epidemiologically exhibits the characteristics of a highly virulent communicable disease. They suggest that these infections were not reduced because of certain inherent deficiencies in present methods of obstetric, pediatric and nursing care of the newborn — the modern mass care of newborn babies in open nurseries may even promote the rapid spread of infection.

Outbreaks occurred in 1940 in three widely separated English cities with a case mortality of 29 per cent. No specific etiology could be discovered, but Ormiston[22] believed that the cause might be a virus or systemic infection or a general metabolic disturbance leading to the proliferation of any pathogenic or potentially pathogenic organism that happens to be present in the bowel.

Epidemic diarrhea occurred in San Francisco in 1942 and 1943, and Geiger and Sappington[23] reported 324 cases with 45 deaths. Although no specific etiology was found there was marked overcrowding in almost every nursery, as well as lowered standards of nursing care.

Weymuller, Beck and Ittner[24] describe an outbreak of epidemic diarrhea involving 19 infants with no deaths. A very carefully thought out prophylactic technic had been in successful operation from 1937 to 1945, when, because of a shortage of nurses, the technic was necessarily violated, and the authors believe that the epidemic was the result.

Rubenstein and Foley[25] reviewed nineteen outbreaks of epidemic diarrhea of the newborn of unknown etiology reported to the Massachusetts Department of Public Health from 1935 to 1945. In all, 258 cases were reported, and 85 patients died. Their investigations revealed almost unbelievable breaks in nursing and formula-making technic. Although the practice was in direct violation of existing regulations, 7 nurseries used a common rectal thermometer. The thermometers were supposedly sterilized by soaking in a bichloride or alcohol solution. Bacterial counts on samples of one of these thermometer dips revealed 7,000,000 viable microorganisms per cubic centimeter, mostly *Esch. coli*. A sample of dip from another nursery revealed 44,000 organisms, the majority being *Esch. coli*, *Proteus vulgaris* and *Staph. albus*. Several nurseries used common oil bottles instead of the recommended individual cups. In many nurseries nurses dip their hands in so-called "sterilizing" solutions. Bacterial

counts on two of these dips, one bichloride and one alcohol, revealed 75,000 and 150,000 organisms per cubic centimeter, including *Staph. aureus*, *Esch. coli*, alpha streptococcus, Lancefield Group D streptococcus and *B. subtilis*.

Adequate space was available in only 3 nurseries, and the nursery staffs were considered adequate in only 3. In 6 of the 10 hospitals formula preparation was not adequately supervised. Samples of formulas from several nurseries gave bacterial counts of 9500, 32,000 and 396,000 per cubic centimeter, the identified organisms being alpha streptococcus, Lancefield Group D streptococcus, *Esch. coli*, *Staph. albus*, *Pr. vulgaris* and *B. subtilis*. Examinations of nipples revealed the presence of large numbers of organisms. One supposedly sterile nipple and one funnel produced a pure culture of *Staph. aureus*.

Cummings[9] and his study group made similar dismal discoveries regarding nursery practice in Michigan. In investigating a number of outbreaks of neonatal diarrhea with stool cultures that were negative for the usual pathogenic organisms, Cummings became interested in the variety of organisms that were identified and he began to speculate on their etiologic significance. The bacteria isolated included coagulase-positive staphylococci, hemolytic streptococci, various strains of Proteus, *Shigella alkalescens*, *Bacterium alcaligenes* (*Alkaligenes faecalis*) and various coliform organisms. These organisms were also routinely recovered from the various supplies, equipment and solutions of the nurseries. In one nursery *Bact. alcaligenes* was isolated from twenty-four stool specimens, fifteen meconium specimens, one infant nose speculum, three nurse stool specimens and three nurse's-aide stool specimens. It was also isolated from nursery basins, enema solutions, subcutaneous saline solutions, soap solutions, table-tops and handbrushes. The conclusion was as follows:

> It has gradually become the opinion of the study group that this type of outbreak is due to massive bacterial contamination with organisms usually considered to be of low or absent pathogenicity together with a complete absence of the usual nursery techniques. *It is felt that filth in the nursery is the principal contributing factor.*

From Rochester, New York, comes a report by Lembcke, Quinlivan and Orchard[26] of two outbreaks of epidemic diarrhea of the newborn that occurred within a four-month period of 1942. The epidemics were controlled by studies on the mode of transmission. The first outbreak affected 28 of the total of 50 infants, with 3 deaths, and was believed to have been transmitted by contaminated rubber nipples and formulas and to a lesser extent by indirect contact. The nurseries were understaffed, babies were diapered before being fed, the hand washing was very perfunctory in cold or tepid water, with very little soap. The rubber nipples were boiled for less than twenty minutes, with some of the nipples floating on the surface, and formulas were inadequately sterilized. Four formula cultures and twelve nipple cultures revealed organisms of the colon bacillus group, presumably of fecal origin. In the second epidemic breast-fed infants were chiefly attacked. Again, infants were diapered prior to feeding, and there was faulty technic in both hand and breast washing.

It is thus readily seen that the reported outbreaks of epidemic diarrhea of the newborn in which no etiology could be established can also be subdivided into two groups. The first consists of outbreaks in nurseries with excellent technics where careful investigation of the affected patients and of their environment by both bacteriologic and epidemiologic studies fail to reveal an etiologic agent; in these outbreaks the negative evidence suggests a virus etiology. In the second and by far the largest group, investigations by trained epidemiologists and bacteriologists failed to reveal a specific organism, but such atrocious nursery and formula-room technic was uncovered that infection with anything was possible. As suggested above the epidemic may even have resulted from massive bacterial contamination with organisms of low or absent pathogenicity.

Prevention

It is evident that diarrhea of the newborn can be caused by a variety of infections. When the services of trained epidemiologists and bacteriologists with adequate laboratory facilities are available, investigation of an epidemic may quickly reveal a specific etiologic agent. When these facilities are not available or if available are not called on, an epidemic may well pass without etiologic diagnosis. Recorded experiences have reported *Pseudomonas aeruginosa*, *Esch. coli mutabile*, hemolytic *Esch. coli*, organisms of the Salmonella group, *K. pneumoniae*, *S. dysenteriae* (Sonne) and Lancefield Group D streptococcus as having been the probable cause of certain epidemics of newborn diarrhea. From the point of view of prevention the presence of these organisms indicates a breakdown in nursery technic that permits carrier-fecal-oral infection of the newborn.

There can be little doubt that virus infection can produce epidemic diarrhea in the newborn. There is suggestive evidence that certain viruses, presumed to be the agent of widespread disease in the community, may also be the cause of coexisting epidemic diarrhea in the local nurseries. There is positive evidence that five epidemics have been caused by a specific virus. In other epidemics there is suggestive evidence for a specific virus etiology in the tendency of diarrhea to spread in the pediatric wards after the baby from a nursery epidemic is admitted for treatment, in contrast to the lack of spread when the infant's infecting organism belongs to the Salmonella or dysentery group. In the absence of a practical method of demonstrating the presence of

a virus in a given epidemic it is impossible to know how many or how few epidemics now classed as of unknown etiology are of specific virus etiology.

The discovery of a practical method to identify and study the specific virus of epidemic diarrhea would be one of the greatest advances in modern pediatrics. It is quite apparent that such a discovery would be of inestimable value in controlling the occasional outbreak of this disease in the obstetric nursery of healthy infants. What is not so generally appreciated is that this disease is not confined to newborn nurseries and that it is endemic in infants' hospitals, where it constitutes a great and perennial problem, the solution of which may well hinge on the discovery of such a method of study.

The etiologic importance of a specific virus in certain outbreaks cannot be overemphasized but at the same time it is well to point out that before negative evidence of a virus etiology can be considered, the epidemiologic and bacteriologic investigations must satisfy the highest standards. Whenever trained public-health officials have investigated numerous epidemics of supposedly unknown etiology, whether the investigations took place in Massachusetts, Michigan or New York, the investigators have uncovered such unbelievably poor nursery and formula-making technic that it has been unnecessary to fall back on an unknown virus to explain the epidemic.

Physicians who have the responsibility of caring for newborn infants in the large and small hospitals throughout the land must set about preventing epidemics of diarrhea whose causes are known. Care of the newborn must be altered so that a baby is not fed a formula containing 396,000 bacteria per cubic centimeter from a nipple covered with a pure culture of staphylococci with hands bearing Salmonella. The nursery staff must be as alert to the dangers of diarrhea in the newborn as the obstetric staff is to the dangers of the hemolytic streptococcus infection in the mother. No nurse with scarlet fever would think of going into the delivery room, and yet a colleague investigating an outbreak of diarrhea in a distant city found a nurse who stated that just prior to the outbreak she had had such a severe diarrhea that she had been forced to spend more time out of the nursery than in it. Perhaps, when these glaring breaks in technic have been controlled, the protection thus afforded may well block the spread of virus infection as well.

The long-term protection of mothers and newborn infants in lying-in institutions demands an extensive national program of new hospital construction. In some hospitals in this country mothers are placed on cots in corridors, and the average stay in these hospitals for mother and child is forty-eight hours. In either new construction or the remodeling of old hospitals primary attention must be given to the needs of the newborn infant. Ideally, each infant should have a private room, or a mother and her infant should share the same room. Certainly, the massing of infants in one nursery should be avoided. The present compromise suggested is that the maximum number of babies assigned to one nursery unit should be the number from six to twelve that can be attended by one nurse.

The following basic principles underlying the control of infection in newborn nurseries that must be satisfied by a successful nursery technic are relatively simple:

Block air-borne infection, both that acquired at short range by droplet transmission and that acquired from pathogenic organisms diffusely distributed throughout the air.

The requirement that each infant in the nursery be provided with 300 cubic feet of air space, or 30 square feet of floor space,[27] is an attempt to protect the infant from air-borne infection by the dilution factor. Many existing hospitals would find it a physical impossibility to satisfy this requirement. The same dilution factor could be provided an infant in half the floor space if the air-circulation rate were doubled. Chapple[28] has reported that recent experience has demonstrated that the air in the nurseries can be sterilized by ultraviolet radiation and by the evaporation of propylene glycol to a strength of 1:5,000,000 in the air (a continuous evaporation of approximately 0.01 cc. of prophylene glycol per cubic foot in a still nursery). The bacterial count in the air can be reduced by the use of an oil film on the floor and by oil-impregnated blankets and linen. It should be apparent that 30 square feet per infant should not be accepted as a fixed requirement when other solutions may be even more efficient.

The control of droplet infection is a more difficult matter; many believe that the ordinary mask is most inefficient. If masks are to be relied on they should be of the deflection type or made of thick layers of nonabsorbent cotton.

For nurseries with limited available space, for isolation nurseries and for premature-infant nurseries an incubator of the Chapple type may be the solution. In this incubator the infant is surrounded by conditioned fresh air drawn directly from outdoors and is further protected from all droplet infection through being completely inclosed in the incubator.

Block infection acquired from the ingestion of pathogenic organisms contained in food or fluid.

This problem is the easiest of all to solve through the terminal unit sterilization of the formula or fluid in the bottle, with the nipple attached and covered.

Block infections acquired from physical contact between the infant and infected personnel or material.

Cummings[9] and his group were able to prevent this type of infection through expert nursing supervision, adequate nursing personnel, infection control among nursery personnel and mothers, carrier control among nurses and mothers, isolation of infected infants, hand technic, sterile supplies and visitor control.

One physician should be appointed as chief of the nursery service with the responsibility and authority for maintaining the standards for the care of all newborn infants, public and private.

It is fortunate that each hospital is not forced to work out for itself the details of standards and recommendations for the nursery care of their full-term and premature infants. The Children's Bureau, in co-operation with an advisory committee of obstetricians and pediatricians and with the help of the Committee on the Fetus and Newborn Infant of the American Academy of Pediatrics, have prepared and published standards and recommendations[27] that, if adopted so far as possible by every hospital attempting infant care, would satisfy the basic principles mentioned and go a long way toward removing the hazard of epidemic diarrhea in the newborn.

All who have been given the grave responsibility of the newborn nurseries in lying-in institutions know that sooner or later, for one reason or another, one or more of their infants may develop diarrhea. The entire literature is in agreement regarding how this situation should be handled.[3, 15, 21, 22, 24, 29] If 1 infant develops diarrhea he should be instantly isolated in an isolation nursery until the significance of the situation can be judged. Should 2 infants from the same nursery develop diarrhea, both babies must be isolated and the nursery quarantined until all contacts have been discharged and the nursery cleaned and made ready for new admissions. With the declaration of the quarantine a mobile nursery should be set up in an available room into which all freshly delivered patients shall be admitted. If 3 infants from the same nursery develop diarrhea the syndrome of epidemic diarrhea of the newborn is assumed to be present, and the situation instantly reported to the board of health.

Strict adherence to this program may well limit a potential epidemic to 1 or 2 cases. The literature stresses the fact that it is disastrous to temporize with this situation.

Should a real outbreak of epidemic diarrhea occur, reported experience insists that the only way to stop the outbreak is to shut down the entire obstetric unit to all admissions until all patients are discharged.

Summary

The emergence of epidemic diarrhea in the newborn as a public-health problem coincides with the transfer of the scene of childbirth from the home to the hospital. Hospitals have been unprepared for this migration, and the mass crowding of infants in nurseries, in addition to the shortage of personnel, has produced conditions favorable for the spread of infection. There is a national need for more obstetric beds and for more nurseries, each to be limited to 8 to 12 newborn infants.

A specific virus etiology for five epidemics has been proved. This virus is probably the cause of many other outbreaks. There is great need for a practical method of demonstrating a possible virus etiology during the progress of an epidemic of diarrhea. It is dangerous to assume a virus etiology for an epidemic on the basis of negative evidence unless the epidemiologic and bacteriologic investigation has been conducted by experts. Specific measures to control or prevent virus diarrhea can be expected to follow the discovery of practical methods to identify and study the virus.

Many epidemics of diarrhea in the newborn have been traced to a variety of pathogenic organisms that could only reach the infant by the fecal-oral route. In the greatest number of epidemics the patients have had none of the usual bacterial organisms in the stools, and yet in a large percentage of these outbreaks trained investigators have found evidence of gross breaks in nursery technic.

Without waiting for the results on future virus research the first obligation is to block all routes by which the infant can acquire bacterial infection in the nursery through a strict application of known nursery and formula-room technics. It is even conceivable that strict technic may block the spread of virus as well as bacterial organisms.

Many large hospitals caring for thousands of babies each year have demonstrated that a strict nursery and formula-room technic can, in spite of personnel shortages and crowding, protect the newborn population from outbreaks of epidemic diarrhea.

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AN UNUSUAL CASE OF ACUTE INFECTIVE POLYNEURITIS WITH VISCERAL LESIONS*

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THE terms "acute febrile polyneuritis," "acute infectious polyneuritis," "infectious neuronitis" and "Guillain–Barré syndrome" are commonly used to designate a class of diseases that are characterized by the acute onset of an ascending flaccid motor paralysis, with loss of tendon reflexes and sensory changes in the extremities, and by elevation of the spinal-fluid protein without pleocytosis. Facial diplegia occurs in some cases and, less often, bulbar paralysis. Paralysis of respiratory muscles results in death in 10 to 40 per cent of cases, and recovery is usually complete in the survivors. The cause of the disease is unknown. Although attempts to isolate a filterable virus or a bacterial neurotoxin have been unsuccessful, an infectious etiology has been predicated because of the usual antecedent respiratory or gastrointestinal infection, the acute febrile course and the nature of the pathological changes.

At the Boston City Hospital we have encountered 3 or 4 cases of acute infective polyneuritis each year. The clinical features of 26 of these cases was the subject of a recent review by Forster, Brown and Merritt.[1] Owing to the high mortality (42 per cent) an opportunity has been afforded to conduct post-mortem examinations in several of them. One in particular excited our interest because of the association of polyneuritis and jaundice. It is reported in detail because of the rarity of such cases in the medical literature.

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Case Reports

CASE 1. The patient (B.C.H. A44-164), a 28-year-old man, entered the hospital on February 2, 1944, because of dyspnea, orthopnea, cough and ankle edema. These symptoms had developed gradually for the previous several years and progressed in the preceding few weeks to the point where he was incapacitated for work.

At the age of 12 years, the patient had had arthritis involving both ankles and causing a deformity of the feet. In 1936, when he was 20 years of age, the deformity had been corrected by a transplantation of the extensor tendons. At that time a cardiac murmur was discovered, and for the next year he was treated by continuous bed rest for suspected rheumatic fever.

Physical examination revealed the patient to be somewhat distressed by flatulence, which was only partially relieved by frequent eructations of gas. He was orthopneic. The heart was moderately enlarged, and there was a systolic thrill over the apex. Diastolic and systolic murmurs were heard at both the base and the apex of the heart. The apical systolic murmur was harsh and blowing, and the diastolic murmur was low, rumbling and presystolic in time. The liver and spleen were not palpable.

The temperature was 98.6°F., the pulse 88, and the respirations 30. The blood pressure was 148/50.

Examination of the blood disclosed a hemoglobin of 86 per cent and a white-cell count of 16,800 that fell to normal thereafter. The differential count was within normal limits. The corrected sedimentation rate was 0.45 mm. per minute. The blood nonprotein nitrogen was 65 mg. per 100 cc. on admission and later 36 and 31 mg. The urine had a specific gravity of 1.032; the first three specimens gave a +++ test for albumin and the sediment contained a few red and white cells, but later specimens were normal. The vital capacity was 1.9 liters. Four electrocardiograms revealed lengthened PR interval of 0.30 to 0.38; the T waves were low, with a slightly high origin, in Lead 1 and diphasic in Leads 2, 3 and 4. These records were interpreted as "consistent with active myocardial involvement."

The patient was digitalized, and all the symptoms disappeared. He was discharged on February 25, with instructions to continue taking digitalis.

On the 2nd day after discharge, he developed a mild discomfort in the epigastrium that rapidly increased in severity, radiating to both sides of the chest anteriorly. The pain was not affected by breathing, by food, or by belladonna. Repeated gaseous eructations afforded some relief. The patient was readmitted on February 28.

Physical examination showed that the patient was considerably distressed by the abdominal pain. He was oriented and rational. There were numerous small, nontender axillary and inguinal lymph nodes. The heart was moderately enlarged, and the murmurs were unchanged. No signs of cardiac decompensation were present. The liver and spleen were not palpable. Neurologic examination revealed no abnormal findings.

The temperature was 98°F., the pulse 100, and the blood pressure 135/40.

Examination of the blood revealed a hemoglobin of 88 per cent and a white-cell count of 6450, with 82 per cent neutrophils, 9 per cent lymphocytes and 10 per cent monocytes. The blood Hinton reaction was negative. The blood nonprotein nitrogen was 50 mg. per 100 cc. on entry and later normal. Examination of the urine was negative. An electrocardiogram showed sinoauricular tachycardia, incomplete auriculoventricular block and a further alteration of T waves — all consistent with a diagnosis of active myocardial involvement. Blood cultures were sterile.

During the first 5 days in the hospital, the abdominal pain became much less severe. The surgical consultant was of the opinion that there were no signs of peritoneal irritation or intestinal obstruction. On the 6th hospital day, the patient had a "sore throat," but the temperature was normal. The white-cell count at that time was 30,200, with 60.5 per cent neutrophils (52.5 per cent adult and 8 per cent band forms), 0.5 per cent eosinophils, 16 per cent small lymphocytes, 9 per cent atypical lymphocytes, 1 per cent large lymphocytes, 9 per cent young lymphocytes and 4 per cent monocytes. The blood smear was thought to be consistent with a diagnosis of infectious mononucleosis. The corrected sedimentation rate was 0.8 mm. per minute. Tests for heterophil antibodies were negative at that time and on two subsequent occasions.

On the 12th hospital day the patient began to have a sensation of tingling like "pins and needles" in the feet and hands and a numbness of the trunk. When the toes touched the foot of the bed, they felt "as if charged with electricity." On the 14th hospital day, he became slightly jaundiced. At that time, the icteric index was 25, the cephalin-flocculation test ++++, and the prothrombin time 60 per cent. In succeeding days, the paresthesias continued, and profound muscle weakness appeared. The neurologic consultant noted mental confusion, a peripheral type of facial weakness on the right side, a flaccid weakness of all four extremities, with the

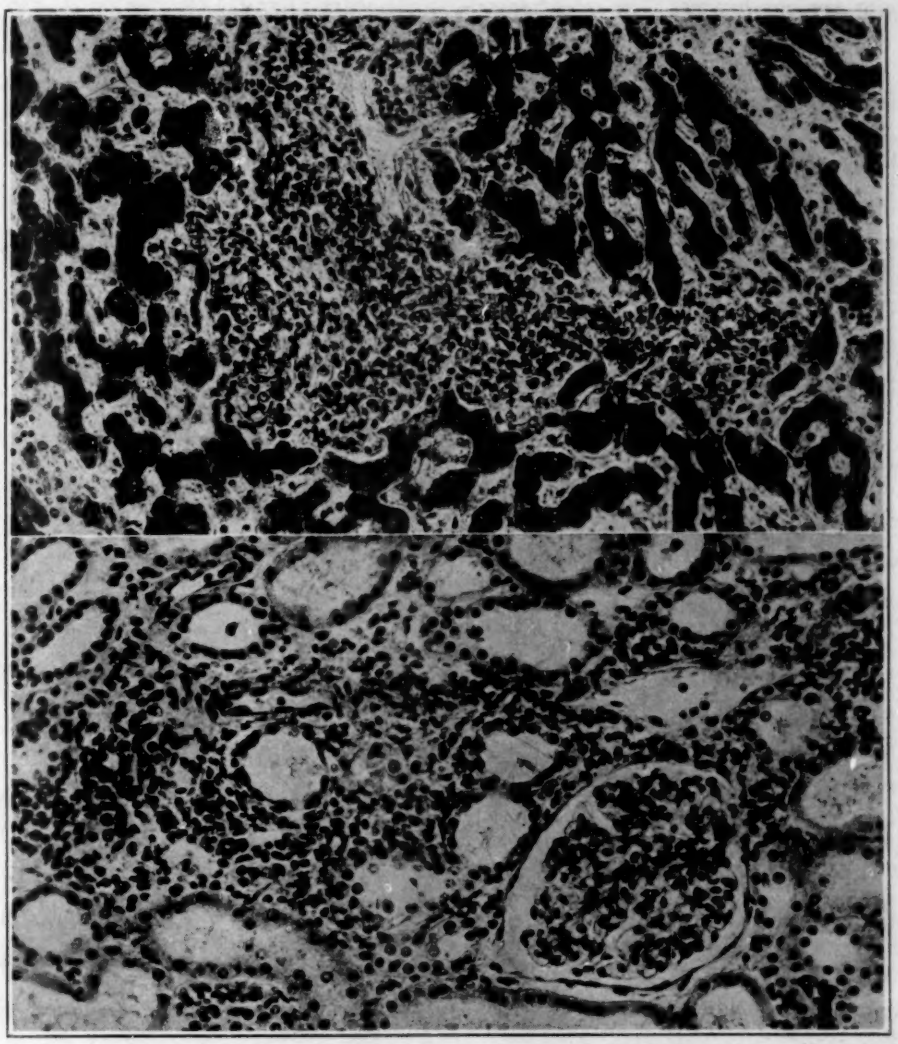

FIGURE 1. A *Shows a Phloxine-Methylene Blue Stain of Portal Area of Liver to Demonstrate Infiltration of Lymphocytes, Plasma Cells and Unidentified Mononuclear Cells.* B *Illustrates a Phloxine-Methylene Blue Stain of the Kidney Showing Interstitial Infiltration of Lymphocytes, Plasma Cells and Rare Eosinophils.*

legs affected more than the arms, loss of tendon reflexes, but active abdominal and flexor plantar reflexes, exquisite muscle tenderness and impairment of all forms of sensation in the distal parts of the lower extremities. On the night of the 16th hospital day, the patient had a "nightmare" and was incontinent of urine. The icteric index was 10; the carbon dioxide combining power was 39 milliequiv. per liter, and the blood sugar 123 and the blood nonprotein nitrogen 63 mg. per 100 cc. The white-cell count was 35,100, with 59 per cent adult neutrophils and 16.5 per cent band forms, 0.5 per cent basophils, 7.5 per cent small lymphocytes, 2.5 per cent atypical lymphocytes, 1.5 per cent large lymphocytes, 4.5 per cent young lymphocytes, 4.0 per cent monocytes, 1.5 per cent young monocytes, 2.0 per cent young plasma cells and 0.5 per cent metamyelocytes. The cerebrospinal-fluid pressure was normal; the fluid was slightly yellow; it contained 20 mononuclear cells per cubic millimeter; the sugar was 54 mg., and the protein 112 mg. per 100 cc.; the Wassermann test was negative. Several brief, generalized convulsions occurred on the 17th hospital day, and during some of these the radial pulse disappeared. A few minutes after a convulsion respirations suddenly ceased at a time when the pulse was quite strong. Death occurred on the 17th day in the hospital.

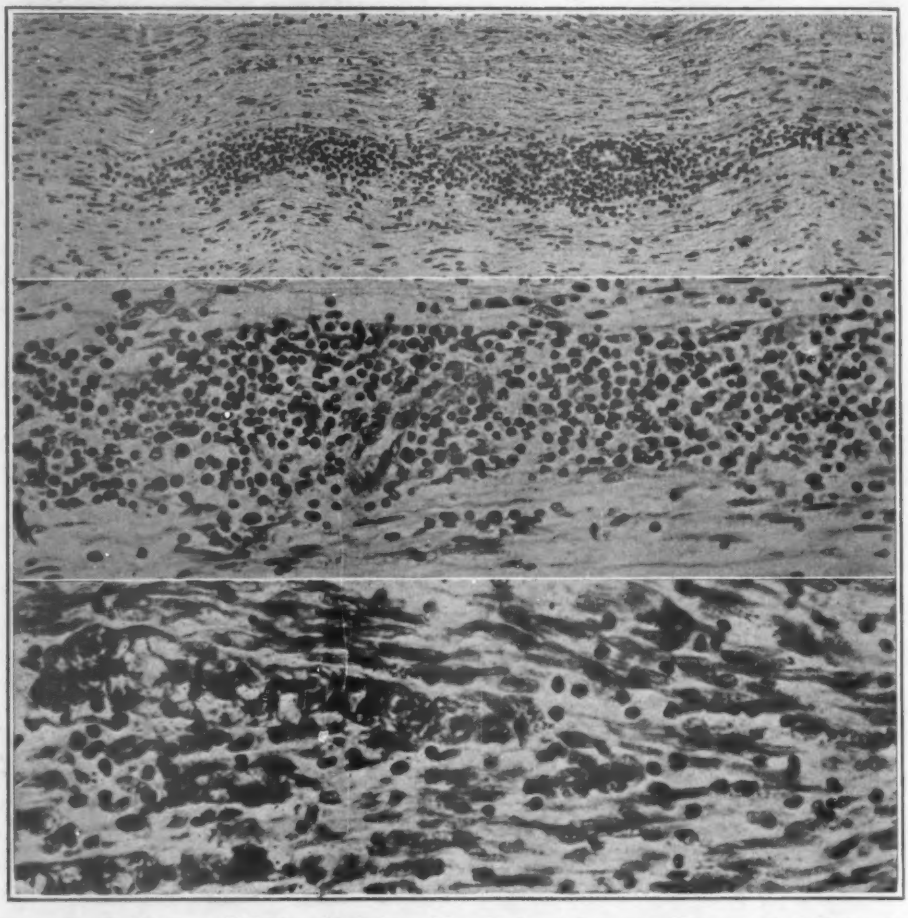

FIGURE 2. A *Shows a Phloxine-Methylene Blue Stain of Peroneal Nerve, with Cellular Infiltration, and* B *a Higher Magnification of the Same Lesion to Demonstrate the Type of Infiltrating Cells.* C *Shows a Sudan III Stain of the Same Nerve. (Note the Swollen, Vacuolated and Fragmented Medullated Nerve Fibers.)*

Autopsy. External examination of the body revealed nothing of note except for slight jaundice of the scleras and skin.

The heart weighed 640 gm. The left auricle was dilated to about twice its normal capacity; the endocardium varied from pale to yellowish white and was calcified in two places. Both leaflets of the mitral valve were greatly thickened, and the anterior one was calcified. The chordae tendineae were shortened and thickened, as were the papillary muscles. The capacity of the left ventricle was twice normal, and the left ventricular wall was symmetrically hypertrophied. The endocardium was smooth except for an area of roughening and calcification, 2.5 cm. in diameter, beneath the aortic valve. The cusps of the aortic valve were thickened and partially calcified, and their free edges were rolled and shortened. There was slight widening of the commissures between the right and left posterior cusps. The coronary ostia and arteries were patent. In the first portion of the aorta, for a distance of about 3.5 cm., the intima was uneven and discolored. Elsewhere in the aorta subintimal streaks of atheroma were visible. The tricuspid valve measured 11.5 cm., the pulmonary valve 7.7 cm., the mitral valve 10.2 cm. and the aortic valve 7.7 cm. in circumference; the right ventricle was 0.6 cm., and the left 1.7 cm. in thickness.

The spleen weighed 580 gm. The surface was smooth and purplish gray. The cut surface was firm and purplish-red, and the malpighian corpuscles were well demarcated. The pulp was firm, and very little could be removed by scraping.

The liver weighed 1680 gm. The surface was smooth and reddish brown. The cut surface was firm, and the lobules were well demarcated by pink periportal tissue. The central veins could not be seen. The gall bladder and bile ducts were normal.

There was a generalized lymph-node enlargement. The nodes in the mesentery averaged about 2.0 cm. in diameter. The largest mediastinal lymph node measured 2.5 by 2 by 1 cm., and the periaortic nodes were 4 by 4 by 1 cm. One lymph node adjacent to the common bile duct measured

4 by 2 by 1 cm. but did not obstruct the flow of bile. There were five discrete lymph nodes along the hepatic duct. The axillary and inguinal nodes averaged 1.0 by 0.4 by 0.4 cm. The cut surface of all these nodes was firm, homogeneous and pinkish white.

The brain weighed 1380 gm. The leptomeninges of the brain and spinal cord were thin and transparent. There were no gross abnormalities of the brain, spinal cord or peripheral nerves.

Microscopical examination disclosed that there was an infiltration of plasma cells, lymphocytes, a few polymorphonuclear leukocytes and some large mononuclear cells both around and within the nerves of the epicardium. A few cells of the same types were scattered through the fat tissue. The myocardium showed a patchy interstitial infiltration of similar types of cells, with more polymorphonuclear leukocytes present than in the epicardium. In addition, there were occasional perivascular lesions consistent with Aschoff bodies. The endocardium contained a focal infiltration of cells of the same types seen in the myocardium and epicardium.

Both the mitral and aortic valves showed a marked increase in collagen and vascularization.

The upper and lower lobes of the right lung and the upper lobe of the left lung revealed an interstitial infiltration of a moderate number of plasma cells, some lymphocytes and immature mononuclear cells of an undetermined type. The alveoli of these lobes were free from exudate except for the presence of a few "heart-failure" cells in some alveoli. The alveoli of the left lower lobe contained numerous polymorphonuclear leukocytes, some large mononuclear cells, many of which contained phagocyted cells, and occasional immature mononuclear cells with a basophilic cytoplasm, type of cell undetermined. The alveolar capillaries contained polymorphonuclear leukocytes, some plasma cells, some immature mononuclears as described above and mast cells. The bronchioles contained an exudate similar to that in the alveoli. The alveolar walls were thickened and congested, and some showed swelling of the alveolar lining cells. The arterioles revealed hyperplastic changes, and an occasional one a necrotizing process. In a large artery there were subendothelial lipoid cells and an infiltration of plasma cells and immature mononuclear cells. Stains for bacteria showed gram-positive diplococci in the polymorphonuclear leukocytes in the alveolar and bronchiolar exudates.

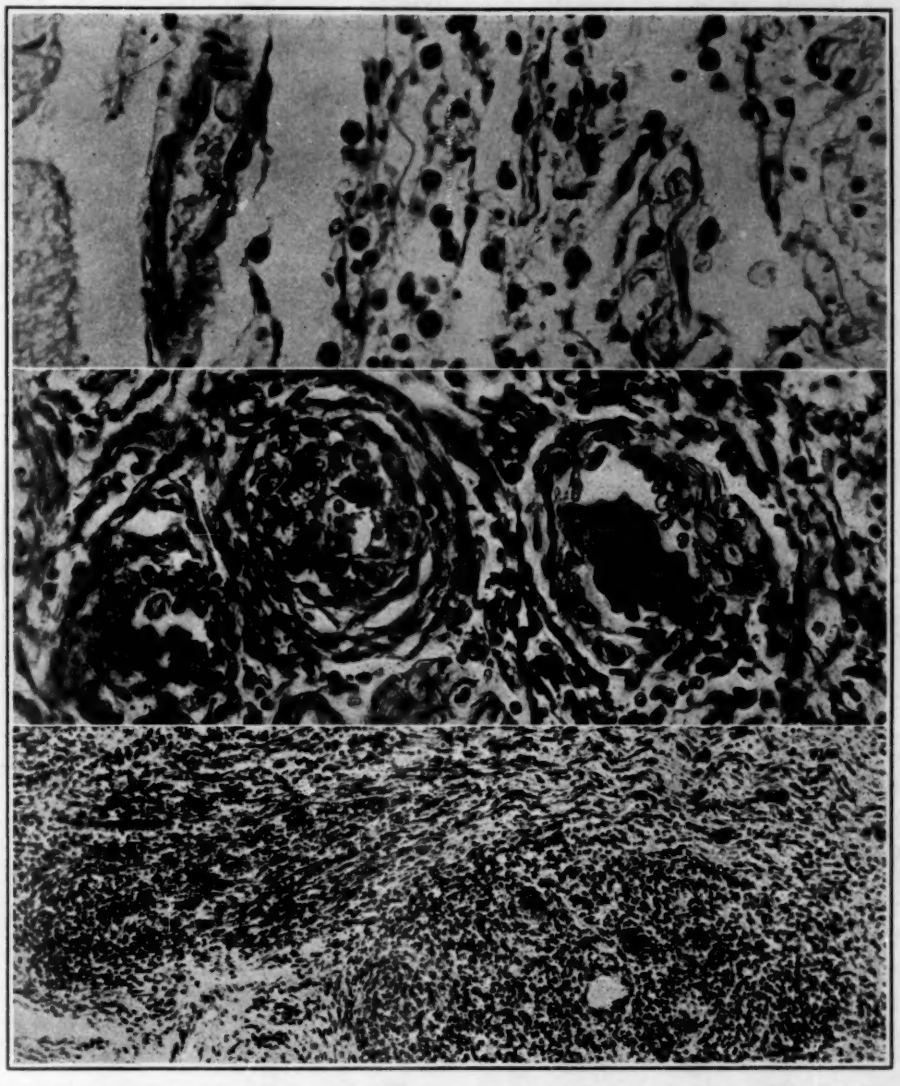

FIGURE 3. A *Illustrates a Phloxine-Methylene Blue Stain of the Spinal Leptomeninges, Showing Cellular Infiltration,* B *a Cajal Silver Stain of a Spinal Ganglion (One Nerve Cell is Shrunken and Pyknotic, and the Other Two are Disintegrated), and* C *a Sudan II Stain of a Spinal Ganglion to Show Cellular Infiltration and Nerve-Cell Degeneration.*

The submucosa of the trachea was infiltrated with plasma cells.

Some of the malpighian corpuscles of the spleen were small and inactive. Others had been replaced by plasma cells, histiocytes and occasional giant cells of the Langhans type. The endothelium of the central arterioles in such follicles was swollen. The pulp was congested and contained numerous plasma cells and polymorphonuclear leukocytes.

The ganglions and the nerves of the stomach were infiltrated with cells as described elsewhere. The muscularis showed an interstitial infiltration.

There was an interstitial infiltration of some plasma cells, lymphocytes, large mononuclear cells and a rare eosinophil in the pancreas. A small ganglion disclosed lesions similar to those described elsewhere.

The liver cells in the centers of the lobules had disappeared, and in the spaces formerly occupied by them were plasma cells, polymorphonuclear leukocytes and large mononuclears, some of which contained pigment. An occasional necrotic liver cell invaded by leukocytes was present. A considerable number of bile capillaries were distended with bile. The liver cells showed a moderate degree of fatty and hydropic degeneration. The portal areas were infiltrated with numerous plasma cells, polymorphonuc.ear leukocytes, occasional eosinophils and large mononuclear cells (Fig. 1*A*).

There was a focal infiltration of plasma cells, lymphocytes and a rare eosinophil in the interstitial tissue of the kidneys (Fig. 1*B*). A stain for fat showed none to be present.

Sections from the lymph nodes from various regions showed essentially the same picture. Their structure was well preserved. The germinal centers were inconspicuous and inactive. The sinuses contained large mononuclear cells, some of which had phagocyted red cells. In the sinuses there were also a few immature cells. The surrounding connective tissue was infiltrated with plasma cells and lymphocytes.

In sections from the bone marrow the granulocytic series were numerous, with normal maturation. An unusual number of eosinophilic myelocytes were present. Cells of the red-cell series were markedly decreased in number. The megakaryocytes were not remarkable. In the sections from the vertebral and sternal marrow were several focal lesions made up of histiocytes and giant cells of the Langhans type.

There was an interstitial infiltration of plasma cells and lymphocytes in the salivary gland.

The pituitary body showed a perivascular infiltration of plasma cells, lymphocytes and large mononuclears in the posterior lobe.

Sections of the peripheral nerves, dorsal and ventral lumbar roots, spinal ganglions, spinal cord, medulla, cerebellum, mid-brain, thalamus and cerebral cortex were stained

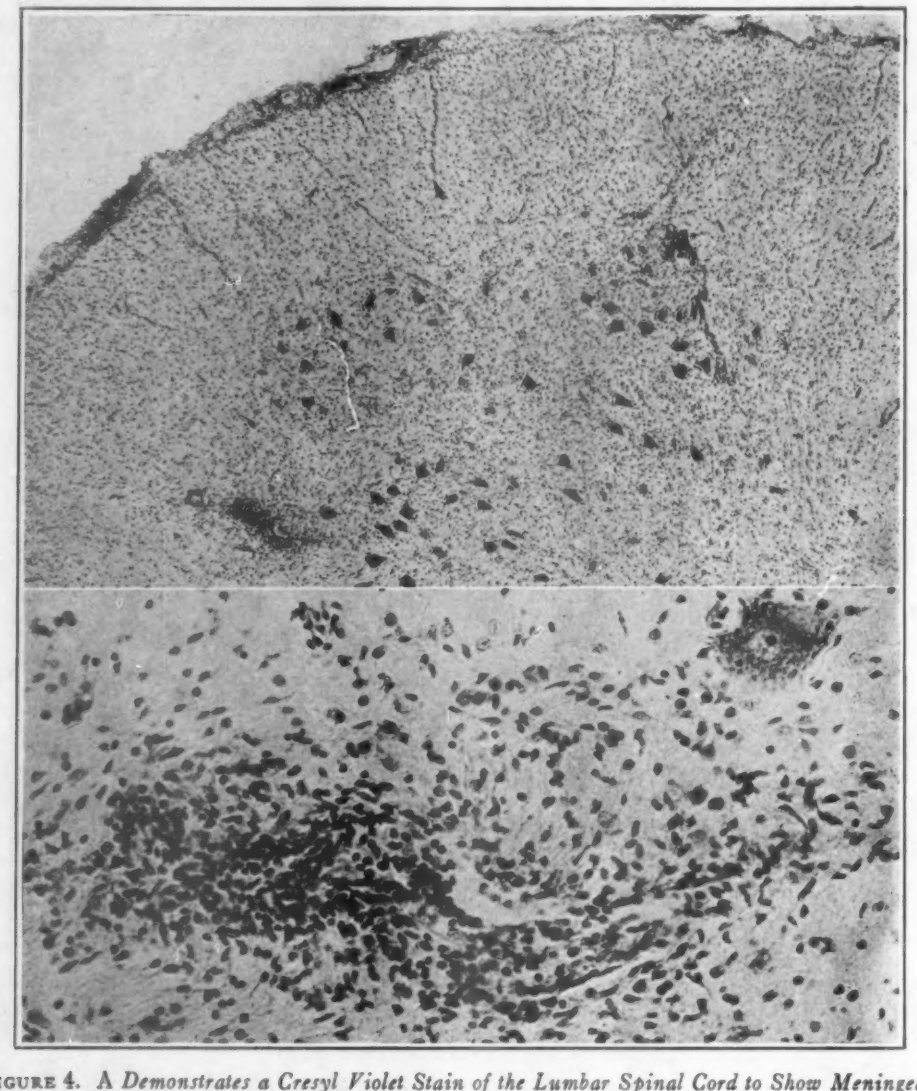

FIGURE 4. A *Demonstrates a Cresyl Violet Stain of the Lumbar Spinal Cord to Show Meningeal and Focal Infiltrations, and* B *a Higher Magnification Showing One of the Lesions in the Gray Matter (the Nodule Consists of Lymphocytes, Plasma and Mononuclear Cells Mixed with Microglial Cells and Astrocytes).*

with cresyl violet. The nerves and ganglions were also stained with ORO for fat, hematoxylin for myelin and silver for axis cylinders.

All the nerves, even the most peripheral twigs, were infiltrated with large aggregates of lymphocytes and plasma cells, unidentified mononuclear cells and a few polymorphonuclear leukocytes; histiocytes were also increased in number. These cells were in the endoneurium, separating the nerve fibers, and had also invaded the perineurium. The spinal sensory ganglion cells and sympathetic ganglions in the bowel and bladder were similarly affected. Most of the ganglion cells had disappeared and were replaced by clumps of satellite cells and phagocytes. Scattered nerve fibers had degenerated, and round and oval-shaped remnants of destroyed myelin remained. In only a few places had these myelin products been phagocytized by histiocytes. The majority of myelinated fibers were intact. The spinal roots were less involved than the nerves. The spinal and cerebral meninges contained infiltrations of lymphocytes, plasma cells and mononuclear cells, and similar cells extended into the posterior horn via the entering root fibers. There were several clumps of microglial cells and lymphocytes and mononuclear elements in the white matter, especially around blood vessels. Some of the anterior-horn cells were swollen and had undergone chromatolysis. A few small blood vessels in the brain and spinal cord were surrounded by lymphocytes and plasma cells (Figs. 2, 3 and 4).

Cultures of the lungs, other organs and blood were not taken. Attempts to culture the lymph nodes and the spleen on chick embryos were unsuccessful, since the material was contaminated with an organism of the *Escherichia coli* group.

The anatomic diagnoses were acute infective polyneuritis, with extensive involvement of the peripheral nerves, spinal roots and sensory and sympathetic ganglions; acute interstitial myocarditis, hepatitis and nephritis; rheumatic heart disease, healed and active; stenosis and insufficiency of the mitral and aortic valves; and bronchopneumonia of the left lower lobe.

In the following case the patient recovered after a few weeks, and hence the diagnosis was not verified pathologically, but the resemblance to Case 1 was so close that the clinical details are added to emphasize the greater frequency and nonfatal character of some cases.

Case 2.* A 46-year-old man had been well until 3 weeks previously, when he became feverish and tired and voided dark brown urine. He improved after 3 or 4 days, returning to work, but malaise continued. Two weeks before admission he became nauseated, and jaundice was noted, deepening during the next week, when numbness and tingling of the fingers and, soon afterward, of the toes and feet set in. The paresthesias rapidly extended up the legs and were accompanied by muscular weakness of such severity that the patient could not stand or walk. There had been a 10-pound weight loss.

Notable items in the past history were rheumatic fever at the age of 17 years and the absence of exposure to a jaundiced person, to metals, to transfusion or to inoculation.

Physical examination disclosed a mentally alert and cooperative patient. The skin was moderately jaundiced. There was a slight enlargement of all palpable lymph nodes. The heart was enlarged, the border of cardiac dullness extending 10 cm. to the left of the midsternal line, the second pulmonic sound was louder than the aortic, and a Grade III rasping systolic and a low-pitched, rumbling diastolic murmur were heard at the apex. The liver had an increased vertical diameter, the lower edge being 5 or 6 cm. below the costal margin at the midclavicular line. The spleen was not palpable. There was no facial weakness or other disturbance of the cranial nerves. The muscles of the arms and legs were partially paralyzed. The legs were more affected than the arms, and the proximal muscles slightly more than the distal ones. The muscles were tender and flaccid. All tendon reflexes, including the plantar, were absent, and the abdominal reflexes were retained. Vibratory and position senses were reduced below the knees and in the hands. Touch sensation was questionably impaired, and other forms of cutaneous sensation were preserved.

The temperature was 98°F., the pulse 88, and the respirations 20. The blood pressure was 120/60.

Examination of the blood revealed a hemoglobin of 105 per cent; and a white-cell count of 12,400, with 83 per cent neutrophilic leukocytes, 8 per cent lymphocytes, 8 per cent monocytes and 1 per cent eosinophils. The nonprotein nitrogen and fasting blood sugar were normal. The cerebrospinal fluid was under normal pressure and, except for a total protein of 80 mg. per 100 cc., was normal. Tests for porphyrins (Watson method) were negative. Heterophil-antibody agglutinations were positive in a dilution of 1:64. The bilirubin was 3.85 mg. per 100 cc., and the icteric index was more than 100. The cephalin-flocculation test was ++++. The total blood protein was 7.7 gm. per 100 cc., with 4.1 gm. of albumin and 3.6 gm. of globulin. The prothrombin time was 76 per cent. The urine contained urobilinogen in dilutions of 1:128 and gave a ++ test for bile, but no albumin or cells were observed in the sediment. An electrocardiogram was normal.

During the 1st week in the hospital the degree of motor and sensory paralysis increased, even though the jaundice lessened perceptibly. The patient became unable to lift his legs from the bed or to sit up without using his arms. All forms of sensation, including pain and touch, were impaired below the knees and elbows. Pain and burning paresthesias of the feet were experienced. The total protein in the cerebrospinal fluid rose to 143 mg. per 100 cc., and there were 7 lymphocytes per cubic millimeter. The treatment consisted of bed rest, an ample intake of carbohydrate, protein and vitamins and intravenous aminoacids.

Throughout the following month the jaundice gradually disappeared, and strength and sensation slowly returned to the limbs. At the end of 3 months the patient had almost completely recovered. The only residual neurologic sign was absent ankle reflexes. The liver function was much improved; the cephalin-flocculation test was +, and the icteric index 10. The last spinal-fluid protein during the 8th week of the illness was still 68 mg. per 100 cc.

The final diagnoses were acute infective polyneuritis; acute interstitial hepatitis; and rheumatic heart disease with cardiac enlargement, mitral stenosis and regurgitation, compensated (Grade I).

Discussion

Case 1 presented several puzzling features. With the development of "sore throat" and the abnormal lymphocytes in the circulating blood a diagnosis of acute infectious mononucleosis was entertained. However, the repeatedly negative tests for heterophil antibodies failed to substantiate this possibility. Then, when jaundice appeared and reduced liver function was demonstrated, the obvious diagnosis was acute infectious hepatitis, which would also have explained the abnormal lymphocytes in the blood smear. When, however, neurologic symptoms became manifest, the diagnosis was again unsettled. We knew of examples of both infectious mononucleosis and infectious hepatitis that were complicated by involvement of the nervous system but could not recall a case of an acute symmetrical polyneuritis associated with either of these diseases.

Post-mortem examination clarified some but not all of the peculiar clinical findings. The generalized weakness, dyspnea and dependent edema, the flatulence and abdominal distress and the albuminuria and slightly elevated nonprotein nitrogen were due to healed rheumatic heart disease and congestive heart failure. The finding of Aschoff bodies in the myocardium provided an explanation

*This patient was observed in one of the medical wards of the Boston City Hospital while the report on Case 1 was being prepared.

for the abnormalities in the electrocardiogram. The upper abdominal pain could hardly have been caused by congestion of the liver, since it occurred when the heart disease was quite well compensated; a more likely possibility was enlargement of the abdominal lymph nodes or of the liver occurring in the terminal illness. The jaundice and impaired liver function were ascribed to the acute interstitial hepatitis or to a combination of hepatitis and central hemorrhagic necrosis. The atypical lymphocytes in the peripheral blood and the leukocytosis were probably part of an infectious disease that mobilized lymphocytes and other mononuclear cells for the extensive cellular infiltrations found in all the nerves and visceral organs. The paresthesias and sensory impairment and the ascending paralysis and reflex loss were manifestations of the acute polyneuritis. The cellular infiltrates in the spinal and cerebral leptomeninges and in the roots were the source of the cells in the cerebrospinal fluid. The convulsions may have been due to heart block (Adams–Stokes syndrome) or the leptomeningitis, and the foci of cellular infiltration in the brain and mental confusion may have had a similar basis. Death was due to the polyneuritis, which finally produced respiratory paralysis, the pneumonia being a contributing factor.

The pathological findings in this case were in general similar to those of the class of diseases called acute infective polyneuritis, the characteristics of which are widespread but uneven lymphocytic, plasma-cell and mononuclear infiltrations of the peripheral nerves, somatic and autonomic spinal ganglions and spinal nerve roots*; irregular degeneration of myelinated peripheral-nerve fibers; varying degrees of change in the nerve cells of the spinal and autonomic ganglions; and swelling and central chromatolysis (axonal reaction) of the motor cells in the anterior and lateral horns of gray matter in the spinal cord. However, Case 1 differed in that the destruction of ganglion cells was much more extensive and the leptomeninges were infiltrated with lymphocytes, plasma and mononuclear cells — a reaction that occurs infrequently in acute infective polyneuritis. Despite these differences, some of which may be only quantitative, it seems to us that the most acceptable classification is in the group of diseases called acute infective polyneuritis. Whether the latter is a single pathologic entity or a pathologic reaction occurring in several different diseases has not been determined.

The histologic changes in the lymph nodes were not those described by Gall and Stout[2] in infectious mononucleosis. According to these authors, the most striking changes are seen in the germinal follicles and the extrafollicular lymphoid tissue. The germinal centers are hyperplastic and contain immature cells, presumably stem cells, and histiocytes, many of which contain phagocyted material. Mitotic figures are numerous. The extrafollicular lymphoid tissue, both cortical and medullary, contains large cells with basophilic cytoplasm. These cells they believe to be identical with the large mononuclear cells in the circulating blood, which are considered characteristic of infectious mononucleosis. Sections of the lymph nodes in Case 1 showed small, inactive follicles and none of the cells in the lymphoid tissue seen in infectious mononucleosis.

Nor were the lesions in the liver similar to those observed in the acute form of epidemic hepatitis described by Wood[3] and by Lucké and Mallory.[4] In acute epidemic hepatitis the destructive process is extensive, and usually all the liver cells are affected, although at times a narrow zone of liver cells persists at the periphery of the lobule. Furthermore, there are a proliferation of small bile ducts and an extensive cellular infiltration of the portal areas with large mononuclear cells, lymphocytes and plasma cells and some polymorphonuclear leukocytes and eosinophils. In Case 1 the liver showed a late stage of central necrosis, with the greater portion of each lobule unaffected, and there was no proliferation of the bile ducts. The cellular infiltrates in the portal areas were similar to those in acute epidemic hepatitis, but these are not characteristic of any one disease, such cellular infiltrations being seen in varied types of infection. The lack of hyperplasia and necrosis in the lymph nodes and spleen must be regarded as further evidence against epidemic hepatitis.

In short, then, autopsy in Case 1 showed lesions in the liver compatible with those in cardiac failure and an extensive interstitial cellular infiltration in practically every organ examined. The abnormal cells found in the blood stream were similar to the unidentified mononuclear cells seen in the visceral infiltrations. Their nature and histogenesis are obscure.

The association of visceral lesions in fatal cases of acute infective polyneuritis has been recognized by other workers. Lhermitte,[5] in 1929, and Lelong and Bernard,[6] in 1935, reported clinical cases in which acute polyneuritis was combined with icterus or subicterus. Bradford et al.,[7] McIntyre[8] and more recently Sabin and Aring[9] presented cases in which acute infective polyneuritis was accompanied by visceral lesions. These lesions consisted of infiltrations of lymphocytes, plasma cells and mononuclear cells in the liver, adrenal glands, kidneys and heart. Ours is the first case, however, in which there was both clinical and pathological evidence of visceral lesions.

Such a case raises interesting questions regarding the relation between acute infectious mononucleosis acute infective polyneuritis and acute hepatitis

*Without cellular infiltrations, assuming of course that sufficient sections of the peripheral nerves, ganglions and roots are available for study, the pathological diagnosis must remain in doubt. It should be remembered that, on clinical grounds alone, polyneuritis due to toxic and metabolic disorders may closely resemble the acute infective variety.

Until the etiology of one or all of these diseases has been determined this matter cannot be definitely settled. It is our impression, however, that there are important clinical and pathological differences.

Unfortunately, no systematic attempt was made to culture a bacterium or virus in Case 1. The pathological findings support the proposition submitted by Sabin and Aring that acute infective polyneuritis is a widespread systemic disease caused by a virus or bacterial toxin that involves the peripheral nervous system predominantly but may produce extensive visceral lesions of such severity as to become clinically manifest in exceptional cases.

Summary

An unusual case of acute infective polyneuritis with jaundice and atypical lymphocytes in the peripheral blood and pleocytosis in the cerebrospinal fluid is reported. The pathological changes consisted in lymphocytic, plasma and mononuclear-cell infiltrations of the peripheral nerves, ganglions and leptomeninges; widespread degeneration of the larger nerve fibers in the peripheral nerves and spinal roots; destruction of the nerve cells in the spinal and sympathetic ganglions; and interstitial infiltration of the liver, kidneys, heart and other viscera with cells similar to those in the nerves.

A similar case, in which the patient recovered and the diagnosis was therefore not verified pathologically, is presented.

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EMPHYSEMATOUS GASTRITIS*

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THE term "emphysematous gastritis" has been employed to designate an infection characterized by the presence of numerous small gas bubbles throughout the wall of the stomach. The lesion is closely allied to phlegmonous gastritis and to cystic pneumatosis, but has certain distinguishing features. Because this disease is so rare, an additional case report is presented below.

In the first case of this type, described by Fraenkel[1] in 1889, the patient died after several attacks of severe abdominal pain, bloody vomitus and diarrhea. Autopsy disclosed that the wall of the stomach was filled with masses of bacteria and small gas bubbles. Fraenkel named the disease "emphysematous gastritis" — a descriptive term that is not entirely satisfactory, since although it describes the presence of gas, it does not emphasize the element of infection.

A somewhat similar case was reported by Morton and Stabins[2] in 1928. This patient also died, and *Bacillus welchii* (*Clostridium perfringens*) was identified as the causative agent.

In the most recent case, observed by Weens,[3] the patient swallowed several ounces of hydrochloric acid. Severe abdominal pain was followed by bloody vomitus. X-ray films of the stomach showed characteristic gas bubbles throughout the wall. The emphysema gradually diminished, but extensive scar formation followed. The patient finally died of sepsis following a gastrostomy. At autopsy a subdiaphragmatic abscess and multiple liver abscesses containing colon bacilli were found. Weens has summarized the literature, and has found no other cases of true emphysematous gastritis, although emphysematous infections of other viscera, such as the gall bladder, urinary bladder and uterus, have been recorded.

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Diagnosis

The essential diagnostic features are severe abdominal pain, nausea and vomiting of blood, prostration, fever, leukocytosis and a typical x-ray picture. The course is usually progressively downhill with death from sepsis.

The disease must be differentiated from acute phlegmonous gastritis and cystic pneumatosis. Phlegmonous gastritis[4] is frequently ushered in by an alcoholic bout, or follows a chemical poison, such as hydrochloric acid, or trauma, such as esophagoscopy. The clinical course is much the same, but x-ray films do not reveal any intramural gas. The mortality is recorded as about 50 per cent.

Cystic pneumatosis is characterized by the presence of localized masses of gas-filled cysts in the wall of a viscus. It is uncommon in the stomach, although 175 cases of involvement of the small

bowel have been collected by Jackson.[5] The accumulations of gas are asymptomatic and are usually found to be incidental to other lesions at laparotomy. If left alone, they tend to disappear spontaneously. Baumann–Schenker[6] has reported the only case of cystic pneumatosis of the stomach that has been recognized by x-ray study before operation.

Robbins[7] believes, from an x-ray point of view, that all cases in which intramural gas bubbles are demonstrated are variants of cystic pneumatosis. It is our opinion, however, that cystic pneumatosis has only one feature in which it resembles emphysematous gastritis: the presence of gas. But in this respect also there is a variation, for the gas in the cystic pneumatosis is usually found in grape-like clusters of cysts, whereas in emphysematous gastritis, it is scattered throughout the walls of the stomach. Emphysematous gastritis is much more closely related to and should be considered a special variant of phlegmonous gastritis.

Case Report

J. C. (M.G.H. 529108), a 15-year-old girl was admitted to the hospital at 8 a.m. on April 28, 1946, complaining of severe abdominal pain. She had been entirely well until 36 hours before entry. Soon after eating dinner, which included fresh salmon but nothing else unusual, she experienced epigastric distress. The remainder of the family, who had had the same meal, had no unusual symptoms. During the night, epigastric cramps developed and were followed by nausea and vomiting. Shortly after the first attack of vomiting, the bowels began to move and there were five loose movements in the following 12 hours. After several episodes the vomiting ceased but the nausea continued. The abdominal pain continued to increase in severity. It remained in the epigastrium, finally radiating directly behind the sternum. It became steady and then more intense. A physician was called 18 hours before entry. Paregoric was prescribed. Four hours later the patient was seen by a surgical consultant, who found that the abdomen was soft throughout and not distended. Peristalsis was normal. It was believed that she had an acute gastroenteritis that would respond to rest.

Figure 1. *Scout Film of the Left Upper Quadrant of the Abdomen on the Sixth Day of the Disease.*

Multiple small gas bubbles are visible throughout the fundus of the stomach. There is no free gas in the peritoneal cavity. Fluid is present in the left pleural cavity.

Four hours before entry, the pain became much more severe and was somewhat relieved by sitting up in a chair. At that time, 8 mg. of morphine given intramuscularly had no effect upon the pain; the abdomen had become silent and more distended. The patient looked definitely sicker than she had before and felt very ill.

Physical examination showed a rather frail, extremely pale girl who was obviously very sick. The skin was cool and moist. The heart and lungs were normal. The abdomen was markedly distended, rigid, tender throughout and silent. Rectal examination was negative.

Figure 2. *Roentgenogram on the Tenth Day, Showing Multiple Gas Bubbles in the Wall of the Body of the Stomach.*

No areas of mucosal ulceration could be demonstrated.

The temperature was 99.5°F. by rectum, the pulse 125, and the respirations 25. The blood pressure was 72/26.

Examination of the blood disclosed a white-cell count of 39,500, with 96 per cent neutrophils, 2 per cent lymphocytes and 2 per cent monocytes. A urine specimen could not be obtained.

A nasal tube was passed immediately, and to our great amazement a total of 2000 cc. of coffee-grounds liquid was obtained from the stomach. This produced some improvement in the general condition. The abdomen became softer and less tender. The diagnosis of acute hemorrhagic gastroenteritis seemed the most tenable. The patient was immediately given intravenous injections of dextrose in physiologic saline solution, with resulting improvement of the systolic blood pressure, which rose to approximately 100. Eight hours later, she was very toxic and disoriented. Climbing out of bed, she pulled out the intravenous needle. A few minutes thereafter she developed a severe state of shock, and when seen at that time, it appeared that death was imminent. While blood transfusions were being prepared, 250 cc. of plasma was rapidly given. This was followed by 850 cc. of whole blood. At that time, the temperature was 104°F. by

rectum, the pulse 160, and the respirations 25. During the first 24 hours in the hospital the patient received a total of 3800 cc. of physiologic saline solution, plasma and blood intravenously, and maintained a urinary output of 600 cc.

On the following morning she was slightly improved. She was still very toxic and dehydrated, with a silent distended abdomen that was slightly tender throughout. The drainage from the inlying Levin tube was still profuse and coffee ground in nature. She then began to run a septic type of temperature, with a rise to 102°F. every evening and a pulse ranging up to 120. Two days after entry, the gastric drainage had cleared considerably and for the first time showed no gross blood. A hemoglobin (photo) of the gastric contents that were aspirated during that period was 4.8 gm. It was estimated that the amount of blood lost from hemorrhage was therefore at least 1000 cc. Three days after entry, it was possible to start feeding by mouth. The abdomen was still somewhat distended, but peristalsis had returned to normal. Examination of the chest at that time showed no evidence of fluid at the left base, and some edema of the face had developed. Fluid in the chest gradually increased in amount. On the 7th day, a chest tap was done. A total of 360 cc. of straw-colored, slightly cloudy fluid was withdrawn, after which 25,000 units of penicillin was inserted. The chest fluid showed no growth on culture.

At the time of entry, an x-ray film had been taken of the abdomen. This was not remarkable except for the presence of several gas-filled loops of small bowel. Six days after entry, repeat plates were made. At that time, a most remarkable picture was observed. The stomach was seen to be dilated, containing a large quantity of gas. Surrounding the entire fundus of the stomach and extending well down on the antrum were numerous small gas bubbles, which were found on all walls of the stomach, but no evidence of free gas in the peritoneal cavity could be found (Fig. 1). Fluid filled the lower two thirds of the left pleural cavity. The x-ray film superficially suggested a stomach full of masses of food, but we were certain that the stomach was empty of solids and fluid.

With the presence of an increasing amount of fluid in the left side of the chest and disappearance of the edema elsewhere, it was believed that a subdiaphragmatic abscess was developing. The penicillin dosage was increased. Ten days after entry, barium was given by mouth. By a barium meal the air bubbles throughout the fundus were shown to lie within the gastric wall (Fig. 2). No areas of ulceration could be demonstrated in the stomach, and the duodenal cap and loop were normal. There was no evidence of subdiaphragmatic inflammatory change or massive pleural effusion. There was no movement of the left leaf of the diaphragm.

Within the following week the temperature gradually declined, and the patient finally became afebrile about 2½ weeks after entry. Penicillin was discontinued 3 days later. Three weeks after entry, x-ray examination showed the swallowing function to be normal. No disease was noted in the esophagus, stomach or duodenum. The fundal portion of the stomach was displaced downward from the left leaf of the diaphragm, probably owing to inflammatory changes taking place in that region. The left leaf of the diaphragm showed marked limitation of motion. The fluid in the chest had disappeared. Meanwhile, the white-cell count had gradually declined to normal, and the hemoglobin was maintained at 12 gm.

Specific therapy consisted chiefly of penicillin, of which 192,000 units was given intramuscularly daily for the first 5 days. This dosage was then increased, so that approximately 300,000 units was administered daily. Sulfadiazine was given intravenously at entry. A maximum blood level of 4.2 mg. per 100 cc. was obtained. The sulfadiazine was discontinued after the initial dose because of the low urinary output and fear of renal complications. The patient also received large doses of adrenocortical extract during the period in which she was in shock. Large doses of vitamins K, C and B were given intravenously. She received a total of five blood transfusions. Unfortunately, no Amigen was available at the time, but the serum protein was maintained over a level of 5.5 gm. per 100 cc. during the hospital stay.

Culture of the gastric contents shortly after entry showed abundant *Escherichia coli*, a moderate number of nonhemolytic streptococci and a few colonies of *Staphylococcus aureus*. Stool cultures on the first available specimen showed no pathogenic organisms.

During the illness the weight had dropped from 85 to 67 pounds. A month after discharge the patient was entirely asymptomatic and had gained 8 pounds. Repeat x-ray films at that time showed no disease in the chest or stomach. A year later the patient reported that she was in perfect health and had had no further gastric symptoms.

Discussion

The cause of the disease in this patient is not clear. It is probable that she first had an acute gastroenteritis, accompanied by vomiting, which produced rupture of the gastric mucosa. This was followed by profuse bleeding and gastric dilatation and then by an invasive infection of the wall of the stomach with nonspecific organisms. It is possible that gas within the lumen of the stomach was actually forced into the stomach wall as well as through mucosal rents, although the wide distribution suggested the formation of gas bubbles by the invasive bacteria.

Although a mixture of *Esch. coli*, nonhemolytic streptococci and *Staph. aureus* was found in the gastric contents, the exact organisms that invaded the gastric wall were not known. Occasionally, mixed infections of *Esch. coli* and nonhemolytic streptococci will produce gas; thus, gas is observed in infections of the leg, or in subdiaphragmatic abscesses from which these organisms can be grown. It can therefore probably be assumed that this mixed infection was the source of the gas.

The extreme prostration was due primarily to blood loss and secondarily to infection. The blood loss, judging from the hemoglobin level of the gastric contents, was at least 1000 cc. After this blood had been replaced, the signs of sepsis became more apparent. The subdiaphragmatic inflammation gradually subsided without the formation of a definite abscess.

Recovery from infection, we believe, was due chiefly to the rather large doses of penicillin employed. The early administration probably maintained the sterility of the subdiaphragmatic and intrapleural fluid collection. Similarly, control of the intragastric infection prevented the development of late scar formation and gastric deformity.

Summary

To 3 cases of emphysematous gastritis collected from the literature a fourth is added. The other 3 patients died. The patient in the case presented above, after penicillin therapy, recovered without complications.

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WHAT INDUSTRY THINKS ABOUT THE MASSACHUSETTS MEDICAL SOCIETY'S PROGRAM OF MEDICAL CARE FOR THE PEOPLE OF MASSACHUSETTS*

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GRANTING that it is necessary to provide more adequate medical care for our citizens, the question arises, How shall this care be distributed? How shall it be administered, and how shall it be financed? Naturally, the further question also immediately arises, What part should the United States Government have in this program? At the present time several bills regarding medical care are pending before Congress. The most important of them are the Wagner–Murray–Dingell Bill (S. 1606), the Maternity and Child Welfare Act of 1945 (S. 1318) and the Taft Bill (S. 545).

The basic principles of medical care as presented by the Society list two main questions. The first is the provision of adequate medical care for those who cannot afford it. The second is the furnishing of that care for those who can afford it. Very properly, I think, the first principle listed is that medical care by voluntary prepayment plans or by direct payment is the responsibility of the local or state government. Part of the burden of this responsibility may be assumed by charitable agencies, such as the various community funds, and part of it by federal grants to state programs, to be administered by state boards of health. This, I think, is one of the fundamental principles of any medical-care program: that it be administered by the government that is closest to the people — that is, by the state or local government. Because the federal Government has at the present time taken over so many of the fields of taxation it seems essential that the financial aid should come from the federal Government. Its administration, however, should be handled on the local level.

The second principle is correctly stated as medical care of those who are able to purchase it by voluntary prepayment plans or by direct payment, which is the responsibility of the individual. In this field government participation should be purely supplementary and educational, and probably any government assistance in a financial way should be along those lines.

The basic principles also state that the eligibility for receiving benefits aided by federal grants should be determined by the individual states; that in any event the patient should have the free choice of his physician, group of physicians, clinic or hospital from among those participating in any plan, government or voluntary, and that on the other hand the physician, group of physicians, clinic or hospital should have the right to refuse or accept the patient and the individual physician should be free to elect or reject without prejudice his individual participation in any medical-care plan; furthermore, physicians, hospitals and nurses should receive adequate remuneration for their services. These principles are essential if citizens are to have the freedom of choice and the recognition of individual rights that are an inherent part of the system of free enterprise. Industry is strongly opposed to any form of socialized medicine, which is merely one step farther toward actual socialism, and the Wagner–Murray–Dingell Bill, especially Title 2 of this act, is no more or less than compulsory health insurance. Industry is very wary of any compulsory government programs, having been burnt by a good many during the last few years.

The Society's principles are carried out in great part by the prepayment hospital plan, the Blue Cross, which is said to act for about 50 per cent of hospital admissions and after ten years of experience has come to be an accepted and important part of the medical program of the Commonwealth. The Blue Shield, which covers surgical and obstetric care rendered in the hospital for approximately 600,000 persons in the Commonwealth and which soon will cover all medical hospital care and surgical and medical services rendered outside the hospital, very ably complements the Blue Cross.

The third operating program of medical care, that of the United States Veterans Administration, covers approximately 600,000 Massachusetts veterans through all types of medical care. Thus, a large proportion of the five million people in the Commonwealth participate in one or more of these three plans.

From the point of view of industry, the Workmen's Compensation Act provides not only hospital, surgical and medical care for all persons injured in industry or in any commercial enterprise or those who are suffering from an occupational disease incurred in the course of their employment but also certain cash benefits for specific injuries and a weekly payment with a maximum of $25 in addition to $2.50 for each dependent during total incapacity and additional weekly payments during partial incapacity. This program covers approximately one fourth of the population and cost industry in 1946 about thirty million dollars. The gap in medical care for persons engaged in industry concerns those who are unable to work because of

*Presented at the annual meeting of the Massachusetts Medical Society, Boston, May 20, 1947.

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sickness and accidents occurring outside the course of the worker's employment. Through the assistance of the Blue Cross, the Blue Shield and the Veterans Administration, as well as by voluntary sickness and accident plans set up by various industries, this gap is rapidly closing. In 1944 the Advisory Council of the Employment Security Division conducted a survey under the authority of the General Court and found that in establishments employing 20 or more persons, 65.9 per cent were covered by some form of nonoccupational sickness or accident protection.

In 1946 a further investigation by the same agency showed that in establishments employing 20 or more persons, such coverage had increased to 71 per cent, and establishments of less than 20 employees, then surveyed for the first time, disclosed that nearly 64 per cent of the workers had some degree of protection against wage losses due to illness. Thus, this gap is rapidly being closed, through entirely voluntary plans.

The demand has arisen in the same quarters as the demand for socialized medicine that a cash sickness-benefit plan be made compulsory for industry. Rhode Island now has such a plan based on the employment security or state fund system. California has a plan financed through a state fund, to which contributions are made as set by law or through private plans under state supervision. New Jersey is considering a proposal, which to date has not become law, for a cash sickness-benefit plan for industry, to be financed by private operation under state supervision. After a careful study of these systems, the Advisory Council recommended that if such a system were instituted it be a privately operated plan under state supervision with the establishment of minimum standards that would preserve the present plans in operation throughout the state. This, I think, is absolutely essential if such a plan is to be put into effect. Industry, however, is not yet ready to accept a compulsory cash sickness-benefit plan. The Advisory Council in its first report to the General Court in 1945 stated as follows:

> . . . the role of government should be one of added impetus to the establishment and expansion of private plans rather than in direct participation. The remarkable growth of group insurance, Blue Cross hospitalization and prepaid medical care, in addition to the long-established individual health and accident insurance policies, is evidence that in Massachusetts private enterprise is meeting the problem and will continue to assume its responsibility if permitted to do so.

This, I believe, is still the point of view of industry, and I think that it coincides with the basic principles expressed by the Massachusetts Medical Society.

In conclusion, the two principles clearly set forth, which must govern all attempts to provide further medical care for the people of Massachusetts, are that the local or state government should assume the responsibility of providing adequate medical care for those unable to obtain it (a great part of this burden should be furnished by federal grants, but the administration should be purely on the state level) and that medical care for those who are able to purchase it by direct payment, voluntary prepayment plans or payroll deductions is the responsibility of the individual. This may be met through collective-bargaining agreements with his employer if he is employed, through voluntary agreements entered into between employer and employee, through private insurance sickness and accident coverage, through the Blue Cross and the Blue Shield and through benefits granted because of the applicant's status as a veteran. Before any compulsory plans are instituted along these lines, the voluntary plans listed above should have a chance to expand and to meet the responsibility. Furthermore, any plan, whether governmental or voluntary, must contain certain safeguards so that there will be absolute freedom of choice between patient, physician and hospital, and adequate remuneration for those whose services are given under such plans.

Correction. On Page 772, in the fourth line of the last paragraph of the summary of the paper "Syphilitic Primary Atrophy" by Drs. Levin, Trevett and Greenblatt, which appeared in the November 20 issue of the *Journal*, "artificial fever" should be changed to read "fever therapy."

MEDICAL PROGRESS

THE NASAL CARRIER OF BETA-HEMOLYTIC STREPTOCOCCI*

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IN spite of the large number of accumulated data concerning the beta-hemolytic streptococcus and its effect on the human host, we are still largely unable to control its endemic reservoir and potential epidemic spread. As a result of the tragic mortality following the influenza pandemic of 1918, intensive efforts were carried out during World War II, first in Great Britain and later in this country, to devise means of curbing the spread of hemolytic streptococci and other respiratory infections, particularly among military recruits. These studies approached the problem from many angles, including chemotherapeutic,[1, 2] immunologic[3] and environmental means of control.[4] In spite of accurate methods of typing hemolytic streptococci,[5] the brilliant development of a new and potent therapeutic agent such as penicillin and the advances made in the understanding of the importance of dust and air in the transport of respiratory disease,[6–11] no single widely applicable and effective control method was devised on either side of the Atlantic to prevent the spread of epidemic streptococcal pharyngitis and tonsillitis and their unfortunate sequelae.[12–14]

With this recent experience in mind, it seems that the classic public-health measures of detection and isolation or treatment of the source of epidemic infection may in the future have to be invoked more often in training camps, schools and institutionalized populations when streptococcal infection approaches dangerous levels. It is well known that the rate of streptococcal pharyngeal carriers may rise from the normal 5 to 10 per cent to a level of 50 to 60 per cent during the development of an epidemic, and until recently there was no unanimity of opinion concerning the relation of a rising carrier rate to the pathogenesis of an epidemic; frequently, clinical streptococcal disease did not parallel the carrier rate,[15, 16] nor was there always a rise in the pharyngeal carrier rate to serve as a sign of an approaching epidemic.[17] Since mass sulfadiazine prophylaxis failed, owing to the appearance of sulfonamide-resistant strains,[18, 19] control of all the carriers has not been feasible by any method devised to date.

Hamburger and his associates[20] have recently proved the existence of the "dangerous" nasal carrier of beta-hemolytic streptococci, who disseminates at least one hundred times the numbers of organisms from his respiratory tract as patients or carriers with pharyngeal implantation only. The possibility of identifying and controlling the relatively few such carriers offers a new method of preventing streptococcal epidemics and solves many discrepancies in the epidemiologic pattern of streptococcal disease. It is the purpose of this paper to assess the evidence that the nasal carrier transmits the bulk of streptococcal infections of the respiratory tract and to review all the known factors pertaining to this condition.

METHODS USED

Recognition of the importance of nasal carriers occurred more or less as a by-product of quantitative studies of environmental contamination by the Commission on Air Borne Infection under the direction of Dr. O. H. Robertson. In these studies extensive use was made of the conveniently identified and typed hemolytic streptococcus as an indicator organism of respiratory contagion in Army hospital wards and barracks, in the course of studies on the control of air-borne and dust-borne contagion. A number of methods were employed in these studies, many of which, although simple, reflect the efforts made to quantitate the amount of environmental contamination by patients and carriers.

The daily streptococcal output of ward patients was estimated, sterile cloth patches 12.7 by 12.7 cm. being stapled overnight to the bottom sheet of the patient's bed. These were subsequently subcultured by rinsing in broth and plating aliquots, from which counts could be made.[20] Previous studies had demonstrated that the bottom sheet showed the highest degree of streptococcal contamination of any item of the bedding.[21]

The potential nasal streptococcal output of patients and carriers was estimated by means of the standard nose-blow test, in which, after sterilization of the hands, the nose was blown three times into a sterile handkerchief, 12.7 by 12.7 cm. in diameter, which was subsequently subcultured as described above.[22]

The number of streptococci residing on the hands was estimated both before sterilization and after the nose-blow test, by washing of the hands for a constant interval in a standard amount of broth, from which cultures were obtained.[22]

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Cultures were made of clothing, bedding and floor dust, the suction device described by Lemon[23] being used. The entire exposed upper surface of the blankets as made up on the bed was "vacuumized" for a constant interval, and subcultures were made of the dirt trapped in the scrubbing chamber of the device. The outer surfaces of blouses, shirts, trousers and coats were similarly cultured, and measured areas of floor surface underneath or around carriers' beds could be sampled with the same device.

Air cultures utilizing both settling plates and the suction device previously described, as adapted for collection of air-borne bacteria, were made.[23] The suction device has a known efficiency for collection of air-suspended bacteria and virus close to 90 per cent and can sample fairly large volumes of air, from 10 to 30 cubic feet. Pour plates made from the scrubbing fluid in the sampler in suitable dilution can be studied for both total bacteria and air-borne hemolytic streptococci.

Cultures of the pharynx bilaterally in the region between the anterior and posterior tonsillar pillars were made at daily, weekly or monthly intervals as indicated. At the same time nasal cultures were obtained by the use of slightly thinner swabs rubbed over the mucosa of the anterior nares to a depth of not more than 2.5 to 3.0 cm.

All streptococcal cultures from the environment were poured in duplicate blood-agar gentian violet (in a 1:500,000 to 1:750,000 dilution) plates for counting and subsequent grouping and typing by the methods of Swift, Wilson and Lancefield.[5] Nose and throat cultures on plain blood agar were carefully described, and the amounts of streptococcal growth roughly estimated, prior to grouping and typing.

A limited number of carriers and healthy controls were studied for anti-streptolysin "0" titer variations, the method as modified by Rantz and Randall[24] being utilized.

The investigations were carried out over a period of four years (1942-1946), chiefly at Chanute Field, Illinois, Camp Carson, Colorado, Fort Lewis, Washington, and the University of Chicago School of Medicine. The co-operation of the United States Army Medical Department facilities at these posts was of great help, and the work was part of an extensive program of research administered by the Army Epidemiological Board, Preventive Medicine Division, Office of the Surgeon General.

Epidemiology and Characteristics of the Nasal Carrier

Early in the course of these investigations certain basic facts became evident that later led to the nasal carrier studies. Parallel studies in hospital wards and Army barracks early in the war indicated that the rate of bacteriologic and clinical cross infection with a given type of streptococcus could usually be correlated with the predominance of this type in the air or dust from floor and bedclothes.[25, 26] Furthermore, analysis of the rate of air and dust contamination in different portions of a hospital ward or barracks squad room revealed definite foci from which the predominant streptococcal type originated. These marked variations in the degree and type of environmental contamination, however, could not always be related to the numbers of streptococci in the pharynx and saliva of the personnel present. Some patients with clinical pharyngitis and strongly positive throat cultures were obviously rapidly seeding their environment with streptococci, whereas many others equally sick distributed very few streptococci to their bedding, clothing or surroundings. The bedding of ambulatory pharyngeal carriers in the barracks was frequently no more highly contaminated by streptococci than that from beds of noncarriers, and in many cases there was no similarity of types, indicating that these beds had been seeded from some other source.

Eventually, a patient with clinical sinus disease was observed by Hamburger to be a most profuse disperser of streptococci, and routine nasal cultures were then instituted. These soon disclosed that almost all cases or carriers of streptococci with strongly positive nose cultures could be classified as "dangerous" in that they were seeding their environment with tremendous numbers of micro-organisms each day, as measured by any one of the several tests employed. Carriers or patients in whom the nasal cultures were weakly positive usually showed less ability to disseminate streptococci, although they often exceeded the simple pharyngeal carriers in this respect.[20]

Since the phenomenon of dosage is of such importance in the determination of experimental infection, and since the degree of dust and air contamination must reflect the quantitative fluctuations in the sum total of streptococcal excretion by carriers and cases, it is of interest to review the differences in the numbers of hemolytic streptococci dispersed by the nasal carrier in contrast to the simple pharyngeal carrier.

Nasal carriers who were "strongly positive" were found to be capable of blowing out an average of 11,000,000 streptococci by the nose-blow test. At times as many as 1,000,000,000 organisms were expelled, and it should be emphasized that some of these patients showed minimal evidence of coryza or rhinitis. Very few if any streptococci were ever found in the nose blows of pharyngeal carriers with negative nose cultures, and weakly positive nasal carriers gave intermediate results. During induced sneezing and coughing, nasal carriers were found to excrete a significantly larger number of streptococci than the pharyngeal carriers, although no significant differences were found in the salivary content of these bacteria.[22]

The hand washes of 74 pharyngeal patient carriers yielded an average of only 4700 streptococci per test,

whereas 106 nasal patient carriers yielded an average of 790,000 organisms. As might have been expected, sterilization of the hands of these nasal carriers by washing in soap and water followed by a rinse in 70 per cent alcohol was considerably more difficult than that of the pharyngeal carriers. The hands of nasal carriers were resoiled back to their original state by a few nose blows into a sterile handkerchief. Forty per cent of the pharyngeal carriers were noted to have negative hand cultures.[22]

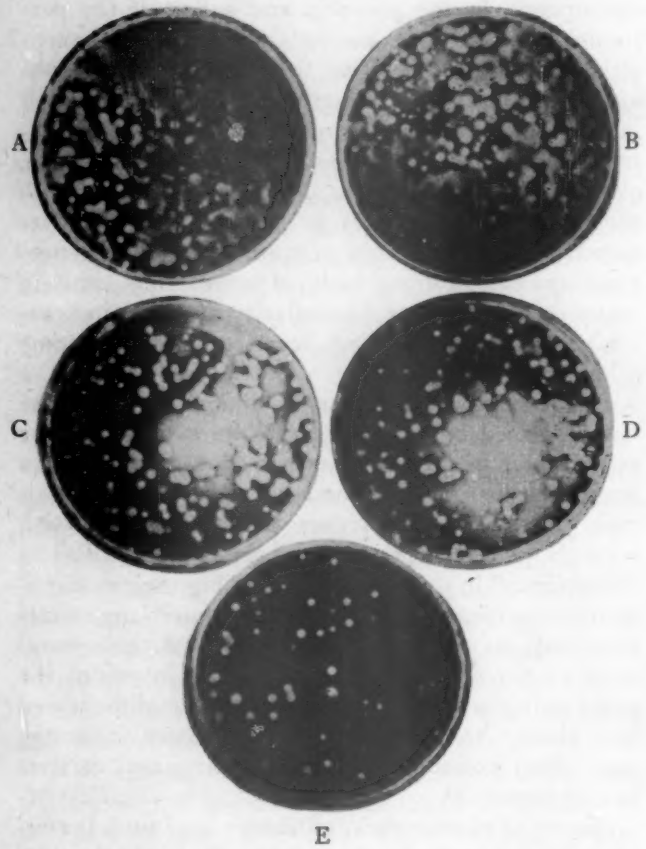

FIGURE 1. *Environmental Cultures, Using a Bubbler-Suction Sampler, from the Vicinity of an Asymptomatic Ambulatory Nasal Carrier (Nose and Throat Cultures Strongly Positive).*

A *demonstrates culture of blankets on bed (75,800 streptococci),* B *culture of comforter (62,100 streptococci),* C *culture of blouse (20,200 streptococci),* D *culture of shirt (17,800 streptococci), and* E *culture of floor dust immediately surrounding and beneath bed (6700 streptococci). The figures represent only a small percentage of the total organisms present; successive samplings yield almost as many streptococci as initial tests. Plates usually represented 1:100 of the total culture.*

The accumulation of streptococci on the cloth patches exposed overnight on the beds of hospitalized patients occurred at a rate about one hundred times as fast in the nasal patient carriers as in the pharyngeal patient carriers. Patients with negative nose cultures usually yielded only 600 to 900 organisms by the patch test, whereas an average of 50,000–60,000 streptococci were recovered from tests on patients with strongly positive nasal cultures; no significant differences were observed between cases with and without a scarlatiniform rash.[20]

Somewhat less marked differences in the blanket content of streptococci were noted in the barracks surveys of beds of ambulatory nose and throat carriers. Both strongly and weakly positive nasal carriers were included in the statistics, and the latter were shown to cause much less contamination of the bedding.[21] Also, no doubt many beds showed evidences of the prolonged survival of streptococci in blankets belonging to men who had once been both pharyngeal and nasal carriers but in whom the nose culture had cleared by the time of the survey. Streptococci have been shown to survive in bedding for periods as long as 5 months.[27] In the barracks, the beds of nasal carriers yielded an average of 66 per cent more streptococci than those of pharyngeal carriers; furthermore, the same type was recovered from nasal carriers and bedding in 74 per cent of cases, as contrasted to only 30 per cent of pharyngeal carriers. The streptococcal content of bedding

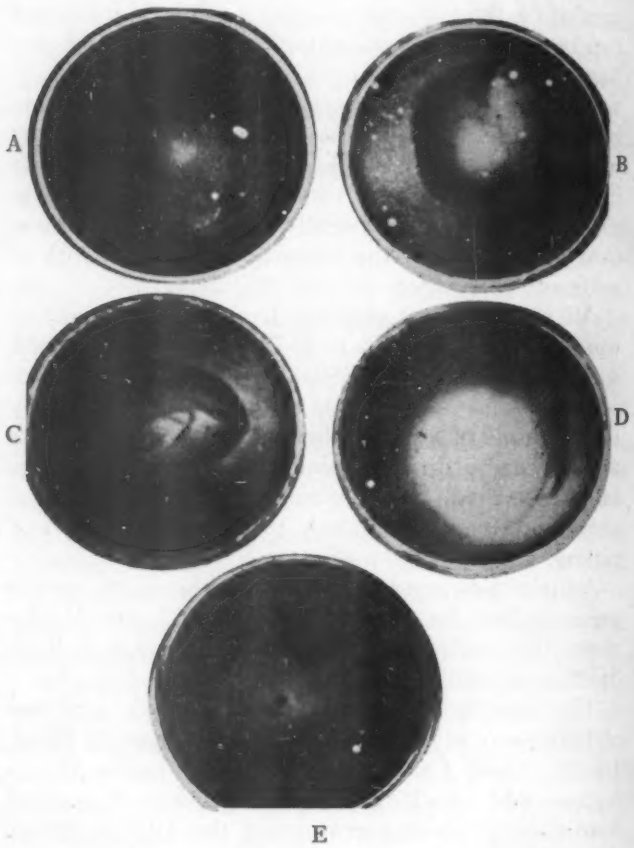

FIGURE 2. *Environmental Cultures, Using Same Technic as That in Figure 1, from the Vicinity of an Asymptomatic Ambulatory Pharyngeal Carrier (Nose Cultures Negative, and Throat Cultures Strongly Positive).*

A *demonstrates culture of blankets on bed (900 streptococci),* B *culture of comforter (500 streptococci),* C *culture of blouse (negative),* D *culture of overcoat (100 streptococci), and* E *culture of floor dust immediately surrounding and beneath bed (100 streptococci).*

belonging to both types of carriers was significantly greater when the same type was recovered from carrier and bed, as might have been expected.

Further studies of the environment of individual nasal and pharyngeal carriers in barracks, in whom the approximate duration of the carrier state was known, demonstrated again the hundredfold difference in amount of streptococcal dissemination to the environment (Fig. 1 and 2), and in several cases the nasal carriers were observed to initiate a localized barracks epidemic caused by their own type of streptococcus.[27–29] It was also found that if the nasal carrier state persisted for four to six weeks, these dispersers could continue to be distinct hazards to their barracks mates, especially if they were transferred to another barracks whose personnel had not been previously exposed to their strain.

The numbers of air-borne streptococci were found to be directly dependent on the presence or absence of nasal carriers of beta-hemolytic streptococci, both in hospital wards and in Army barracks. Over twenty-five times as many streptococci were recovered from the air of a ward housing patients with strongly positive nasal cultures as from the air of a similar ward housing tonsillitis-pharyngitis patients with positive pharyngeal cultures only.[20] In the barracks, whenever large numbers of hemolytic streptococci were recovered from the air (up to 100 to 150 per cubic foot), a nasal disperser of streptococci was identified in the personnel.[29]

The majority of traceable hospital cross infections were found by Hamburger to be due to nasal carriers on the wards. Almost all patients who spread infection had high streptococcal outputs as measured by the patch test (from 40,000 to 500,000 organisms) whereas the majority of patients who failed to spread infection had less than 40,000 streptococci on the most highly contaminated patch. Eleven of 12 cases of ward cross infection were traced to nasal carriers, and only 1 to a pharyngeal carrier. A food-borne streptococcal epidemic was traced to a food handler with a strongly positive nasal culture who handled pies in the incubation period of the pharyngitis; cultures of the hands yielded 10,000,000 organisms of the type responsible for the epidemic.[28] As mentioned above, barracks epidemics were traced to single nasal dispersers among the personnel. One asymptomatic nasal carrier caused 10 cases to be admitted to the hospital from his barracks in two weeks. He was hospitalized for another three weeks for study of the streptococcal output and treated for five days with full doses of sulfadiazine; during his hospital stay he acquired another streptococcal type and was still carrying large numbers of streptococci in his nose on discharge. The new type was not present in any of the personnel of the new barracks to which he was assigned; within a few days he had heavily contaminated the bedding, floor dust and air of both floors of this barracks, and induced an epidemic of streptococcal cross infection involving 30 per cent of the personnel. Four patients who carried the new type of streptococcus were admitted to the hospital from this barracks during the next two-week period. Extensive environmental contamination was found to precede the appearance of cross infections.[29] This patient illustrates the fallacy of present-day quarantine regulations and the role that the hospital itself may play in the dissemination of streptococcal disease among the population it serves.

Finally, the nasal carrier rate of troops was found to correlate well with the rates for streptococcal disease. In units having a low incidence of streptococcal disease (0.4 per cent of personnel hospitalized in a six-week period) the nasal carrier rate was 0.13 per cent, or 1 for every 41 pharyngeal carriers. Units having a moderately high incidence of streptococcal disease (5.7 per cent of personnel hospitalized) showed a nasal carrier rate of 4.2 per cent, or about 1 nasal carrier out of every 4 throat carriers. Under epidemic conditions in which nearly a third of a unit was hospitalized in this period, 40 per cent of the population were found to be nasal carriers, or four fifths of all the throat carriers.[30] These figures can be compared with the incidence of nasal carriers of streptococci among hospital admissions for pharyngitis or scarlet fever, which ranged from a quarter to two thirds of the cases.[20, 31]

In view of the obvious importance of the nasal streptococcal disperser, numerous studies were carried out to define more clearly the factors in the individual patient that determined the presence or absence of a profuse growth of streptococci in the nose. It should be emphasized that almost all nasal carriers yield strongly positive pharyngeal cultures. It appears from routine repeated surveys of populations that the nasal carrier state is a far more transient condition than the pharyngeal carrier state, occurring in the first few days or weeks after the patient has acquired a new streptococcal type. Clinical disease may or may not follow the appearance of streptococci in the nose and throat. Occasionally, streptococci have been found only in the nose and not in the throat,[26, 31] but usually within twenty-four to forty-eight hours when the cultures were repeated the throat was positive. The same type of streptococcus is almost always present in both locations. Proliferation of streptococci in the nose may or may not be associated with coryza or symptoms and signs of paranasal sinusitis.

The majority of so-called "healthy carriers" found by surveys gave a history of recent sore throat, cough or nasal discharge, but signs of tonsillitis and sinusitis were not frequent. Many showed a moderate degree of pharyngeal injection, but no reliable distinction could be made at any time on a clinical basis between this group and the non-carriers. Most of the nasal carriers discovered by cultural surveys did not report to sick call at any time, and the rest were usually returned to duty

by the dispensary physician.[32] In spite of the mildness of the signs and symptoms, evidence was obtained of a rising or elevated antistreptolysin titer in 50 to 70 per cent of the ambulatory nasal carriers, and it was concluded that in most cases they represented missed cases of infection rather than true contact carriers with only a symbiotic disease state. It should be emphasized that some of the most "dangerous" of these carriers complained of few symptoms.

Among patients hospitalized with positive nasal cultures the majority had a follicular tonsillitis or severe pharyngitis, and in this group about 60 per cent of cases showed roentgenologic evidence of sinus disease.[20] The streptococcal output, however, did not depend on the presence or absence of sinus involvement, and one must conclude that in many cases marked proliferation of streptococci occurs on the nasal mucosa alone. It seems likely that the development of immunity to the infecting strain can be fairly well correlated with the decline in streptococcal output of many of these carriers and with the gradual return of the nasal mucosa to its normal streptococcus-free state. Within ten days after admission the streptococcal output of 80 per cent of hospitalized nasal carriers fell to low levels, and two months later only 13 per cent of these patients carried any streptococci in the nose, although half were still pharyngeal carriers. Many barracks carriers showed an even shorter duration of the nasal carrier state.

Since the symptoms of the ambulatory nasal carrier may be mild and nonspecific, however great their streptococcal output, their control assumes corresponding importance and difficulty. Cultural surveys, which are the only sure method of selecting these cases out of the herd, may be performed rapidly by the placing of the streakings of nasal swabs from 4 cases on quadrants of a single blood-agar plate. Two to 4 patients can be cultured a minute, depending on the amount of teamwork. Presumptive identification of these cultures is much easier than that of the pharyngeal cultures, especially when large numbers of organisms are recovered.[28]

The chemotherapeutic control of ambulatory nasal carriers has been temporarily successful both with prophylactic doses of sulfadiazine (in the absence of a large proportion of resistant strains) and with calcium penicillin in oil and beeswax (Romansky formula). Sulfadiazine therapy (1.0 gm. daily or more), which clears the nose in two thirds or more of asymptomatic carriers and even in patients may markedly lower the streptococcal output to a safe level, should be carried out for at least three weeks to prevent relapse in many carriers after cessation of therapy. Calcium penicillin given in one dose of 300,000 units a day in oil and wax was found to be much more effective in eradicating streptococci from both the nose and the throat in patients, but half the cases showed a relapse with the same type after five to seven days of therapy. One quarter of the total again manifested strongly positive nose-blow cultures one to five days after termination of therapy. This period of therapy seems to have been too short for adequate control of patient carriers.[33]

Discussion

The importance of the nasal carrier of hemolytic streptococci has been referred to by numerous workers in the field of streptococcal epidemiology. Among the first to utilize nasal cultures were Meleney and Stevens,[34] who, in 1926, reported an epidemic of postoperative streptococcal wound sepsis that was traced to the instrument nurse, a nasal carrier. There were several other possible sources for infection from pharyngeal carriers on the surgical staff, but careful serologic analysis utilizing reciprocal agglutination and agglutinin absorption confirmed the identity of one of the wound strains and that isolated from the nurse's nose. In 1933 Colebrook[35] was impressed with the possible role of nasal carriers in the production of puerperal sepsis, because of the increased potentialities for contamination of the hands by handkerchiefs, and cites several cases in which nasal carriers seemed to be the source of infection. Hare,[36] in 1941, first demonstrated that nasal carriers expelled more streptococci than throat carriers, noting in 3 cases that the contamination of the person was more widespread and of longer duration than when only the pharynx was involved. In 1944 Coburn[37] reported two outbreaks of streptococcal disease traceable to post-infection carriers who had clinical and roentgenologic evidence of sinus involvement; nose cultures were not performed. Gordon,[38] in 1932, observed that discharged scarlet-fever patients with rhinitis or sinusitis were more likely to transmit infection than patients without complications, or those with other complications, such as suppurative otitis media. The relative lack of infectivity of simple pharyngeal carriers noted above confirms the conclusions of Bloomfield and Felty,[39] who in 1923 observed the paucity of cross infection between chronic pharyngeal carriers and normal persons rooming together.

The finding that the nasal streptococcal carrier is in most cases an "acute" carrier who has only recently acquired the organism and from whom dispersion of streptococci gradually diminishes as local or general immunity develops re-emphasizes the conclusions drawn by Blake[40] after World War I regarding the importance of acutely infected individuals in the genesis of epidemics. Although Bloomfield and Felty's study emphasized the local parasitism of the tonsil by hemolytic streptococci under conditions of sporadic disease, vast and bitter experience has repeatedly shown that virus diseases affecting the upper respiratory tract, especially influenza and measles, predispose to secondary infection with the streptococcus, which then readily

invades on a wide scale the mucosa of the upper and lower respiratory tract.[41] In view of Glover's[42] production of nasal dispersers of streptococci in ferrets doubly infected with influenza A virus and Group C streptococcus, it would be extremely interesting to know what proportion of acute nasal streptococcal carriers have had their respiratory mucosa already damaged by influenza virus or the virus of the common cold. These ferrets proved highly contagious when placed in close association with other healthy ferrets, and it was shown that administration of streptococci alone infected only the tonsils of the animals, but not the nasal membranes. Infection of the membranes with streptococci was possible only by means of an antecedent or concomitant virus infection.

Since the chemotherapeutic control of nasal dispersers by any means except mass prophylaxis presupposes routine carrier surveys at close intervals (ideally, twice weekly), the use of less cumbersome and protracted safeguards appears to be indicated. Hand contamination from the nose seems to be the most important route of dissemination, and much use could therefore be made of oiling procedures for all handkerchiefs, masks, toweling, bed linen, blankets and clothing with which patients and carriers might come in contact. These procedures have been adapted to standard laundry use and are cheap, nonirritant and safe from fire hazard. The treated textiles cannot be distinguished from those untreated, although the process permanently impregnates woolen fabrics.[43, 44] These oiling procedures have been shown to prevent the major portion of secondary contamination of air and dust resulting from streptococcal carriers.[13] Air disinfection with glycol vapors has been shown to enhance the effect of oiled bedding in reducing streptococcal aerial pollution.[14]

As Hamburger[20, 28] suggests, it seems logical to set up new criteria for the discharge of patients with streptococcal tonsillitis from hospitals, regardless of the presence or absence of a rash. No patient should be discharged who has a strongly positive nasal culture, and all such patients should be carefully isolated or given adequate suppressive chemotherapy. Probably, most patients with only a few colonies in the nose cultures could be discharged after three weeks in the hospital, provided that there were no known susceptibles such as young children or patients with rheumatic fever in the home environment. Proper instruction concerning cleanliness of the hands and disposal of nasal secretion would be an additional safeguard. At least two nose cultures prior to discharge should be obtained, preferably at least forty-eight hours or more after discontinuance of chemotherapy.

There is evidence that other bacteria causing upper respiratory infections are disseminated from the nose in a manner similar to that by which the hemolytic streptococcus is spread. For instance, nasal diphtheria has been regarded by Place[45] as "by far the most contagious, as discharge through methods and habits of handling may be readily spread about by contact, handkerchiefs, towels, lavoratories, candy, toys, hands, garments, etc. Moreover, nasal cases very often escape early detection, under the guise of colds, and allow a longer period of contact than other forms." Localized diphtheria epidemics have been traced to nasal carriers of the same type.[46] Ward and Henderson,[47] in 1907, were not able to control a diphtheria epidemic until they had isolated the nasal as well as the pharyngeal carriers among the population.

Similarly, it is thought that the spread of *Staphylococcus aureus* can best be related to its presence or absence on the nasal mucosa, from which the hands are secondarily contaminated. Miles and his coworkers[48] studied the relation of nasal staphylococcal carriers to wound infection and the bacteriology of the carriers' skins, noting carrier rates of 40 to 50 per cent, with many nasal carriers yielding positive skin cultures. It was possible to prove similarity of types in the nose and on the hands by the phage typing technic in a limited series, and the conclusions were that the hands were the main source for dissemination of coagulase-positive staphylococci from the nose.

Contamination of the hands by staphylococci and streptococci from nasal sources may explain some of the epidemics of food-borne disease caused by these organisms reported from military and civilian sources in the past few years.[28, 49] It is well to remember that organisms from the upper respiratory tract of food handlers may be just as dangerous as intestinal pathogens when opportunity occurs for food contamination, and, in addition, much of the intrafamilial spread of beta-hemolytic streptococci may occur by this route.[50] Transmission of streptococci by hand-to-mouth contamination (or via fomites) seems to be particularly important in children. As a result of observations on the inadequate sterilization of mess kits during the influenza pandemic of 1918-1919, Cumming and Spruit[51] stressed the significance of streptococci and pneumococci on the hands and in the wash water in explaining the high incidence of secondary bacterial infection among troops in World War I. They noted beta-hemolytic streptococci in the mess-kit wash water in 84 per cent of examinations and on the hands of 38 per cent of military personnel. Their studies, which included observations on the experimental transmission of mouth-inoculated *Serratia marcescens*, tended to show a lower incidence of pharyngeal streptococcal carriers and a reduced incidence of influenza and measles among troops boiling all their mess gear as contrasted with a carefully selected control group with inadequate wash-water temperatures.[52] It is well to recall these studies to emphasize the varied routes that upper respiratory pathogens may take in passing from person to person.

Finally, it should be remembered that the simple pharyngeal carrier of beta-hemolytic streptococci always represents a hazard to his fellows, different from that arising from a nasal carrier only in degree. Much of the sporadic transfer of streptococci probably takes place chiefly from the pharyngeal carriers, as the very low incidence of nasal carriers under these conditions attests.

Summary

There is at present ample evidence that the "strongly positive" nasal carrier of beta-hemolytic streptococci disperses approximately one hundred times the number of organisms to his environment as the pharyngeal carrier, irrespective of the presence of a rash or the severity of the symptomatology. Most of the nasal carriers represent cases of streptococcal infection in the acute phase so far as can be determined by clinical and immunologic evidence, although in many cases symptoms are so mild that they are overlooked or neglected. Probably less than 10 per cent of these carriers continue to disperse large numbers of streptococci for periods longer than two or three weeks. Such carriers as these, however, may initiate localized outbreaks of streptococcal disease, and when acute nasal carriers are multiplied as a result of cross infection, the incidence of clinical disease will rise sharply, with a secondary rise in the pharyngeal carrier rate representing contact carriers and convalescent cases with persistent parasitism and slight potentiality for production of secondary cases. Widespread and lasting control of the proliferation of streptococci on the nasal mucosa is one of the chief problems in the prevention of epidemic streptococcal disease today.

Chemotherapy with sulfadiazine (in the absence of resistant strains) or penicillin, newer technics of environmental dust control with oil and air sanitation using germicidal vapors have been shown to have definite value in reducing possibilities for cross infection offered by nasal streptococcal dispersers.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 33521

PRESENTATION OF CASE

A fifty-year-old woman was admitted to the hospital because of abdominal pain.

One week before entry, two hours after a normal bowel movement, the patient was suddenly seized with severe persistent lower abdominal crampy pain, and a few hours later she began to vomit. A physician gave her some pills, following which she passed some gas, but the pain persisted throughout the abdomen and she continued to vomit. She was then taken to another hospital, where a Miller–Abbott tube was passed. This lessened the distention, and she stopped vomiting.

Twenty-three years before entry and again three years later, the patient had full-term normal deliveries but had had a great deal of pain throughout both pregnancies. For this reason she was sterilized six years after the last pregnancy by the removal of the tubes and one ovary; the appendix was also removed. The menopause occurred nine months before admission, and she had subsequently had only intermittent bleeding.

Physical examination revealed a slightly distended "doughy" abdomen with active peristalsis. There was a freely movable, nontender golfball-sized mass in the left midabdomen. Vomiting had ceased. She passed gas by rectum, and an enema gave good results.

The temperature was 98°F., the pulse 90, and the respirations 22. The blood pressure was 118 systolic, 70 diastolic.

The red-cell count was 4,660,000, with a hemoglobin of 11.4 gm. and a hematocrit of 39 per cent. The white-cell count was 11,200, with 87 per cent neutrophils. The serum chloride was 85 milliequiv. per liter. The serum protein, the nonprotein nitrogen, the fasting blood sugar and the prothrombin time were within normal limits.

An x-ray film showed the tip of the Miller–Abbott tube in the left midabdomen, with dilated loops of small bowel both proximal and distal to the tip.

The day after admission the dilated loops proximal to the tube were no longer visible, whereas those in the right lower quadrant close to the cecum persisted. On the third day the tip of the Miller–Abbott tube was still in the same position. Injection of 50 cc. of barium through the tube revealed knuckling and narrowing of the small bowel to a width of 1.0 cm. or less. There was no evidence of prominence of the mucosa. The loops of bowel immediately proximal and distal to the tip were not dilated. A film taken four hours later showed the barium to have stopped in the same loops, and the persistent dilated loops of small bowel were noted in the right side of the abdomen.

The patient was treated with Amigen, vitamin B_2, hykinone and intravenous 5 per cent dextrose in saline solution. The chloride increased to 94 milliequiv. per liter, and the hematocrit and hemoglobin were reduced to 34 per cent and 9.4 gm. respectively.

An operation was performed on the third hospital day.

DIFFERENTIAL DIAGNOSIS

DR. CLAUDE E. WELCH: This is an obvious case of intestinal obstruction. One of our major problems is to determine the site of the obstruction. Clinically the best clue is the location of the pain. The patient had severe lower abdominal crampy pain, which usually means that the colon is involved rather than the small bowel. However, when a patient begins to vomit shortly after the attack of cramps, obstruction is presumed to be in the small bowel rather than the large, and the repeated vomiting is evidence more in favor of small-bowel than large-bowel obstruction. Since the character of the pain during the pregnancies is not described I am unable to make much out of this symptom.

"She was sterilized six years after the last pregnancy by the removal of the tubes and one ovary." That, I might suggest, is an unusual method of sterilization and implies that there was something

wrong with the pelvic organs at that time requiring removal of the ovary.

"The menopause occurred nine months before admission, and she had subsequently had only intermittent bleeding." That is a confusing statement, and is impossible to interpret.

One should pay attention to masses, even though they are very small, but because of its character I shall say that this so-called mass was the distended balloon of the Miller–Abbott tube. I may be wrong, but it seems to fit better with the history.

There is no note of vaginal or rectal examinations. In trying to abstract some of these histories it is difficult not to leave out some of the important points, but I think that we are justified in assuming that those examinations were made and were negative.

The blood studies showed evidence of dehydration as brought out in the latter part of the record. There was a slight leukocytosis, with an increase of neutrophils, but not enough to be of great significance. The serum chloride was low because of the repeated vomiting. It would have been interesting to know whether the stool that was obtained by enema was guaiac positive or negative. That specimen probably eluded the watchful eye of the nurse on the ward and was disposed of before it reached the laboratory.

May we see the x-ray films?

Dr. Stanley M. Wyman: The first examination shows dilated loops of small bowel assuming a lace pattern. There is some barium remaining in the colon from a previous examination, and there is gas extending up to the region of the splenic flexure. The film taken the next day shows some decrease in distention of the small bowel, but the tip of the Miller–Abbott tube is not changed in position. The position of the tube is constant throughout the series of films. Two days after the first film barium was introduced into the Miller–Abbott tube, and the spot films taken at the time of examination show the tip of the tube in the apparent area of narrowing at this point in the small bowel. It is rather hard to interpret this without having done the examination. There is no definite evidence of a change in the mucosal pattern. This is a loop of dilated small bowel just distal to the tube, so that there is at least one point of narrowing in the small bowel without apparent involvement of the mucosa.

Dr. Welch: Do you see any evidence of air in the liver radicles?

Dr. Wyman: There is no evidence that I can see of gas extending up into the liver shadow.

Dr. Welch: You can see nothing that suggests a shadow of calcium rather than barium in the lower bowel?

Dr. Wyman: I do not see any stones scattered through the abdomen. I think that there is at least one point of narrowing in the small bowel that does not involve the mucosa, so far as I can tell.

Dr. Welch: The x-ray examination has been very helpful in many respects. Even if he had done the examination, I imagine that Dr. Wyman would agree that it would be difficult to interpret the spot films.

When faced with a case of intestinal obstruction, the surgeon must attempt to be more specific and attempt to name the causative factor of the obstruction. The first question is to determine whether the large bowel or the small bowel is involved. In this case the x-ray examination has been of great value because the barium studies have shown a fairly normal colon with no distention, indicating that the lesion involves the small intestine. The surgeon must next determine whether a strangulating or a nonstrangulating obstruction is present. Strangulating obstruction is identified by the presence of tenderness, by the absence of peristalsis and often by leukocytosis. A patient with this type of obstruction must be operated on promptly. Obviously, a strangulating obstruction was not present in this case, so that we immediately rule out the strangulating lesions of the small bowel producing obstruction, which include intussusception, mesenteric thrombosis and volvulus. Consequently, we pass on to the nonstrangulating types. To begin with, there are a few rather unlikely lesions of the small bowel that can be excluded immediately. It is unlikely that this patient had an inflammatory lesion of the distal ileum. I assume that the statement in the x-ray interpretation that there was evidence of prominence of the mucosa is meant to indicate no evidence of regional ileitis. Is that correct?

Dr. Wyman: I should think so.

Dr. Welch: It would be unusual for regional ileitis to appear with the first symptoms at fifty years of age and then with acute obstruction. The same statement is true of other types of inflammatory diseases in the distal ileum. What, then, are the common and likely causes of the obstruction in this patient? If one can judge by percentage figures, hernia, either obvious or concealed, should be the most obvious choice. A careful examination was made of this woman, and the chances that a hernia was undiscovered are rather slight. A femoral hernia is often hard to find and may be overlooked. However, that is unlikely. In the presence of operative scars, bands or adhesions furnish by far the most frequent causes of intestinal obstruction and, until we can discover a better cause, must be considered seriously.

The next thing to be considered is a foreign body. In this part of the country, where persimmons are not a favorite part of the diet, the only foreign body that commonly obstructs the small bowel is a gallstone. That is a difficult diagnosis to make, especially in the absence of signs pointing to the gall bladder. There was no gas in the biliary radicles and no evidence of stone by x-ray examination.

The third most probable cause to be considered is that of tumor involving the bowel. It is unlikely that this was a primary carcinoma of the ileum. The obstruction came on suddenly, and there was no evidence of bleeding by rectum. Our attention has been called to a mass, and that might be interpreted as tumor of the small bowel. I believe that we have a much better right to call it a distended Miller–Abbott tube. Could this have been one of the peculiar benign tumors of the lower portion of the small bowel, such as carcinoid? Again, there is no evidence in its favor; its rarity rules this diagnosis out as a likely possibility.

Could the patient have had metastatic carcinoma arising somewhere in the abdomen and involving the terminal portion of the small bowel, especially at the multiple points of obstruction suggested by x-ray study? It is possible, but again in a woman the usual source of such a carcinoma is in the ovary and we have the right to assume that the pelvic examination had been negative.

Since we have to make a diagnosis in this case of intestinal obstruction, I believe that it is obstruction of the small bowel, in the lower ileum, nonstrangulating in type, due to bands from the previous operation.

Clinical Diagnosis

Intestinal obstruction.

Dr. Welch's Diagnosis

Intestinal obstruction, due to adhesive bands.

Anatomical Diagnoses

Acute oophoritis and perioophoritis.
Intestinal obstruction, due to inflammatory adhesions.

Pathological Discussion

Dr. Tracy B. Mallory: I am sorry that the surgeon who operated on this woman is not present to discuss the diagnostic problem. So far as the record is concerned he did not commit himself beyond the diagnosis of intestinal obstruction.

The patient was explored, and several coils of small bowel were found adherent to a mass lying at the brim of the pelvis. The adhesions were fibrinous, and it was possible to separate them with blunt dissection. When that was done, it was found that the mass was a considerably enlarged ovary measuring 7 cm. in diameter, the center of which was filled with pus. The ovary was removed, and following operation the patient made a rapid convalescence. Inasmuch as the uterus and both tubes had been removed fourteen years before, I believe that we are safe in assuming that this was a hematogenous infection of the ovary.

CASE 33522

Presentation of Case

First admission. A fifty-one-year-old housewife was admitted to the hospital because of shortness of breath.

Twenty-nine years before entry she had an attack of rheumatic fever characterized by pain and swelling of the wrists and hands. She was put to bed for three months. After this she felt well and had two children. She was told, however, that her heart was weak, and eighteen years before entry her last pregnancy was terminated for this reason. Two years before entry she began to have dyspnea on exertion, requiring bed rest occasionally for a day or two. Recently she had to use two pillows at night. Two weeks before entry she "contracted a cold" with cough and daily expectoration of several tablespoonfuls of foamy, sometimes blood-stained sputum. She became extremely dyspneic and orthopneic, and was treated with digitalis, Mercupurin and penicillin with doubtful improvement. On the night before admission she became worse, with severe cough and orthopnea.

Physical examination revealed an orthopneic woman, with some cyanosis and malar flush. The neck veins were distended and pulsating. There were basal rales on both sides of the chest. An examination of the heart was reported as follows:

> The heart is tremendously enlarged to the left and right, with the point of maximal impulse and left border of dullness at the anterior axillary line in the fifth interspace. There is enlargement to the right in the fifth space, with the right border of dullness 5 cm. from the midsternal line. There are sounds of good quality. The mitral first sound is strong and booming, and the pulmonic second sound is very much increased and duplicated. At the apex there are Grade IV systolic and diastolic murmurs, with an accompanying thrill. The systolic murmur is loud and harsh through all of systole and is transmitted to the back. The diastolic murmur is very loud and low pitched and occupies all of diastole, with the strongest element in middiastole. In the right lower portion of the chest anteriorly, over the upper border of the liver, there is a very loud Grade III or IV rough systolic murmur with a thrill. At the base no murmurs that can be separated from the widely transmitted ones noted above can be made out. The rhythm is totally irregular.

The liver edge was palpated 10 cm. below the costal margin and was firm. The spleen was felt 2 cm. below the costal margin. No abdominal fluid was detected. There was only minimal pitting edema of the ankles.

The temperature was 101.4°F., and the pulse 96. The blood pressure was 120 systolic, 80 diastolic.

An electrocardiogram showed auricular fibrillation, the ventricular rate averaging 100. The QRS complex was 0.09 second, with normal voltage, right-axis deviation, absent R, prominent QS and very low T waves in Lead 1, sagging ST segments in Leads 1, 2 and 3, slightly inverted T waves in Leads 2 and 3, small R and prominent S waves in

Leads CF_4 and CF_5 and upright T waves in Leads CF_2, CF_4 and CF_5. There was one ectopic ventricular beat.

X-ray examination of the chest revealed that the heart was enormously enlarged in all directions, the transverse diameter being 22 cm. and the chest 25.5 cm. There was calcification of the arch of the aorta. The left auricle appeared extremely large, pressing on the left main bronchus and compressing

FIGURE 1.

it posteriorly. There was fluid in both pleural cavities. The hilar shadows were prominent (Fig. 1).

Examination of the blood disclosed a hemoglobin of 14 gm. and a white-cell count of 12,000, with 90 per cent neutrophils. The sedimentation rate was 14 mm. in 1 hour (corrected). The prothrombin time was normal. The van den Bergh reaction was 0.8 mg. per 100 cc. direct and 1.4 mg. indirect. The cephalin-flocculation test was +++ in forty-eight hours. The urine showed a + test for albumin, with an occasional hyaline cast in the sediment. The stools were guaiac negative.

The patient was treated with penicillin, a low-sodium diet, ammonium chloride and Purodigin. She had an episode of severe dyspnea and pain in the right side of the chest the day after admission. Following this her condition improved steadily. The temperature, pulse and respirations were normal, and the chest rales cleared. After two weeks she was discharged to a nursing home on a maintenance dose of Purodigin.

Second admission (five weeks later). The patient was readmitted because of severe dyspnea and chest pain. In the interval she had stayed in bed most of the time. For two weeks prior to readmission she had sharp pains in the right axilla, aggravated by cough and respiration. On the day before readmission she had a "cold," and around supper time, she suddenly developed a persistent cough and foamy, white sputum. The dyspnea and pain in the right side of the chest became very severe, and she had to sit up in bed all night. By morning she felt feverish and had noticed some blood staining of the sputum.

Physical examination revealed dyspnea, cyanosis and cough. The temperature was 103°F. There were rales in both sides of the chest but no signs of consolidation or fluid. There was a transient friction rub in the left axilla. The heart signs were the same as on the previous admission. The rate was irregular with a pulse of 90 and a blood pressure of 115 to 120 systolic, 70 to 80 diastolic. The liver edge was palpated four fingerbreadths below the costal margin and was pulsating. There was shifting dullness in the flanks and bilateral ankle edema, more on the right side. There was no calf tenderness or pain on dorsiflexion.

Examination of the blood disclosed a red-cell count of 4,100,000, with a hemoglobin of 14 gm., and a white-cell count of 8200, with 88 per cent neutrophils. The urine showed a specific gravity of 1.018, with a ++ test for albumin. The sputum was negative for pneumococci. An electrocardiogram was similar to that on the previous admission.

A bilateral femoral-vein ligation was done on the second hospital day. The congestive failure rapidly subsided with digitalis and Mercupurin, and after the fifth hospital day, the temperature remained normal. The patient was discharged at the end of two weeks on a low-sodium diet, digitalis, ammonium chloride and Aminophylline suppositories.

Third admission (five weeks later). The patient returned to the hospital because of an incarcerated femoral hernia. A herniorrhaphy was done, and 13 cm. of infarcted ileum was resected. She made a good recovery and was discharged eighteen days later on a regime of digitalis, ammonium chloride, a low-sodium diet and mercuhydrin intramuscularly.

Fourth admission (six weeks later). Because of the gradual onset of additional symptoms of feverishness, night sweats, shoulder pains and malaise, the patient was readmitted. There were no chest, abdominal or back pains, gross hematuria, chills, pains in the fingertips or petechiae.

The physical examination was essentially similar to that on previous admissions except that the examiner noted systolic murmurs at the base on each side of the sternum, in addition to the apical murmurs and those in the right side of the chest. The spleen was firm, tender and palpable 3 cm. below the costal margin. There was only slight ankle edema.

The temperature was 100°F.

An x-ray film of the chest revealed some fluid in the right pleural sinus. The electrocardiogram was unchanged.

The hemoglobin was reduced to 10 gm. The red-cell count was 3,000,000, and the white-cell count was 7100. A cephalin-flocculation test was +++ in twenty-four and forty-eight hours, and the bromsulfalein test showed only 4 per cent retention of the dye. Repeated blood cultures were negative. The urine gave a + test for albumin.

The patient was hospitalized for sixteen days and during that time the temperature ranged from 98° to 100°F., the pulse from 70 to 80, and the respirations from 20 to 30.

Final admission (eight weeks later). The patient had been reasonably well until the midnight before entry, when she complained to her daughter that she felt as though the blood were rushing to her head, and asked to go to the bathroom. The daughter helped her to the bathroom, and on returning the patient rather suddenly became severely short of breath and cyanotic and was unable to speak. There was no unconsciousness, convulsive movements or incontinence.

Physical examination revealed a cyanotic, dyspneic, slightly confused woman with a temperature of 100°F. The neck veins were distended and pulsating. The heart was fibrillating at a rate of 95 per minute (apical), and the blood pressure was 150 systolic, 90 diastolic. The lungs were resonant throughout. The respiratory sounds were faint, and there were tight expiratory wheezes over the entire chest. There were moist inspiratory rales at both bases, more on the right. The liver edge was palpated three to four fingerbreadths below the costal margin and was firm and sharp. The spleen was not felt. There was a +++ pitting edema of both ankles. There was flaccid paralysis of the right lower part of the face and the right arm and leg, as well as questionable aphasia. The deep tendon reflexes were equal on both sides. Babinski and Chaddock signs were suggestive on the right. The neck was not stiff.

The white-cell count was 7100, with 83 per cent neutrophils. The hemoglobin was 11 gm. The urine showed a ++ test for albumin. The cerebrospinal fluid contained 200 red cells per cubic millimeter in one tube and 500 red cells in the other.

The patient was given oxygen, Cedilanid and mercurzanthin. The next day she developed dullness and almost absent breath sounds in the right middle and lower lobes, and the white-cell count increased to 16,100. An emergency bronchoscopy was done, and this increased the breath sounds and aeration temporarily although an obvious plug of secretion was not removed. Tracheal rales were soon heard over the entire chest. She became exhausted and unresponsive and died quietly two days after admission.

Differential Diagnosis

Dr. Oglesby Paul: May we start by seeing the x-ray films?

Dr. Stanley M. Wyman: The huge heart described is seen to be essentially unchanged from the first observation until six months later. It is enlarged both to the right and to the left; the greater enlargement seems to be toward the right. There is a small quantity of fluid in both pleural spaces. The left oblique view shows the left main bronchus elevated, compressed and narrowed by what I take to be a huge left auricle. The right oblique view again shows a large left auricle. There is calcification of the aortic arch well over to the right. No note is made by fluoroscopy of calcification in the heart itself. I cannot see any on the film. Later films show the fluid in the right pleural space to have increased considerably five months after the first film. There is no very good evidence of enlargement of the region of the left ventricle.

Dr. Paul: It would be very hard to make a diagnosis of pulmonary infarction with a heart of that size, would it not?

Dr. Wyman: Yes.

Dr. Paul: Starting with the first admission, we have no reason to question the fact that this patient had had rheumatic fever in the past leading to severe rheumatic heart disease. The question is, What had changed the course from one of fairly good tolerance of effort to one of sudden appearance of congestive failure? Several things should be considered. She may have developed auricular fibrillation or flutter, or she may have had a reactivation of rheumatic fever or developed some other infection, perhaps even bacterial endocarditis. A third possibility is that of pulmonary infarction. It is interesting that when she came in, she was in severe failure. The murmurs can be interpreted as indicating mitral stenosis and marked mitral regurgitation; the question arises, What was the murmur in the right lower portion of the chest anteriorly over the liver area? That may have been the murmur of mitral regurgitation well heard in the right side of the chest because the left auricle extended so prominently to the right. Ordinarily, I suspect that it is better heard posteriorly and in the axilla than anteriorly. A less likely possibility is that it represented the murmur of aortic stenosis. The splenic enlargement does not necessitate a diagnosis other than rheumatic heart disease and congestive failure. Occasionally, we see patients in congestive failure who do have palpable spleens, without other evidence of bacterial endocarditis. It should raise the possibility of bacterial endocarditis but does not necessitate making that diagnosis.

The electrocardiogram is consistent with mitral stenosis. The absence of anemia on the first admission is of course some evidence against rheumatic activity and bacterial endocarditis. The patient

was considered on the first admission to have infection, not rheumatic in type, for she was given penicillin and did improve. It seems probable in view of the second admission that she actually had repeated episodes of pulmonary infarction, and following ligation of the femoral veins bilaterally she appears to have done well for a time.

The first two admissions may therefore be explained on the basis of severe rheumatic heart disease complicated by multiple pulmonary emboli.

We do not need to go into the third admission, when a herniorrhaphy was performed, except to say that despite marked valvular disease, the patient showed good tolerance for major surgery. Nothing else is indicated in that note to show any new findings.

The fourth admission is interesting. For the first time the patient complained of persistent fever, sweats and general malaise, and for the first time she was anemic. When she was admitted, she showed less evidence of failure than she had on previous admissions, and had no leukocytosis. There were no positive joint findings, although it is stated that there were joint pains, and no nodules, petechiae, clubbing of the fingers or weight loss were mentioned. The pulse was not rapid. It is stated that repeated blood cultures were negative. She was in the hospital for sixteen days, so that there was a considerable period in which cultures could have been taken; apparently, they were negative or showed only contamination.

She was sent home, presumably with a diagnosis of active rheumatic fever. I should think that she would not have been sent home had bacterial endocarditis been seriously considered. No mention of treatment is made, but I gather that there was no other evidence of infection suggesting the need for chemotherapy.

She did reasonably well during the intervening eight weeks. "Reasonably well" is to my mind a tribute to her heart, for with a heart of this size one would hardly expect the patient to do well, especially in view of the fact that in the previous six months, she had had four hospital admissions, two operations and repeated pulmonary emboli. The fact that she did reasonably well suggests that there was no serious infection, particularly bacterial endocarditis. There is no mention of new symptoms.

Finally, she turned up in the hospital for two days with a terminal cerebrovascular accident of some type. Again, at this final admission we are not told that examination showed clubbing or petechiae or enlargement of the spleen. The anemia was, if anything, less than before. (Perhaps the patient was dehydrated.) Certainly, it does not appear that she was more anemic. She finally succumbed with pulmonary complications due to plugging of the right main bronchus.

I believe that the diagnoses until the fourth admission were rheumatic heart disease, mitral stenosis and regurgitation, possibly involvement of the aortic valve, quite possibly a functional tricuspid regurgitation and old pulmonary infarction. The question now arises, Was the fourth admission related to the final admission when she succumbed? It may have been, for she may have had subacute bacterial endocarditis on both occasions. A diagnosis of active rheumatic fever on the fourth admission and then bland embolism on the final admission do not necessarily bear any relation, however. So far as I know, there is no correlation between rheumatic activity and the incidence of fatal or nonfatal emboli from the left or right auricle.

What is in favor of subacute bacterial endocarditis? The history is classic: feverishness, sweats and malaise occurring insidiously, low-grade fever and joint pains (which raise the possibility of associated active rheumatic fever). Joint pains of a vague type are at times described in subacute bacterial endocarditis without other clear evidence of rheumatic activity. The spleen was palpable, and there was a new murmur, as well as a basal systolic murmur not previously mentioned. It is possible that the murmur was a transmission from the Grade IV murmur described at the apex — it was loud enough to have been heard at the base. The absence of leukocytosis is not unusual in bacterial endocarditis. Finally, one could explain the terminal episode as representing the tossing off of an embolus from the vegetations in the heart. There are certain things against that diagnosis, however. The first and most important is that during the eight-week period in which the patient went home following the fourth admission, she apparently did quite well. I believe that if she had had bacterial endocarditis, she would have had a downhill course, at least sufficiently obvious to have deserved reference to symptomatology. I also believe that she would have become more anemic rather than having the blood improve to the extent that it did. Repeated blood cultures were negative. Also on the final admission, the spleen was not palpable and appears to have grown somewhat in size rather than diminished. No weight loss is noted in the history, and there was no clubbing of the fingers. Also somewhat against the diagnosis of bacterial endocarditis is the fact that the patient had marked valvular disease. We more commonly associate bacterial endocarditis with moderate or mild valvular disease than with severe valvular disease. Finally, the combination of auricular fibrillation and bacterial endocarditis in the same patient is not common.

A few remarks about rheumatic fever itself are indicated. She was a known rheumatic patient, and rheumatic activity in an adult is probably more frequent than we appreciate. She did have a low-grade fever, of the type one expects more in rheumatic fever than with subacute bacterial endocarditis. She had joint pains and anemia. She also

improved to some extent in the hospital without chemotherapy, for she was sent home. I would not expect her to have improved significantly on bed rest if she had had bacterial endocarditis. To explain the terminal episode, we must assume that she had an embolus from the left auricle, which was not related to the presence of active rheumatic fever.

On reviewing the evidence I should say that we have more against the diagnosis of bacterial endocarditis than for it. I should like therefore to make a diagnosis of chronic rheumatic heart disease, with mitral regurgitation and mitral stenosis. The patient may have had aortic disease, but I cannot be sure from the evidence at hand. I would not be surprised if a functional tricuspid insufficiency was indicated by the presence of a dilated tricuspid ring, and believe her heart to have been large, with a big left auricle and evidence of old pulmonary infarcts, which may have been found as healed scars at postmortem examination. I suspect that she had a terminal embolus from the left auricle to the left cerebral hemisphere. I think that subacute bacterial endocarditis is less likely and shall put it as my second choice.

Dr. Howard B. Sprague: Would you entertain the possibility of adding an interauricular septal defect to explain the fact that she did so well for so long and also to explain the somewhat abnormal transmission of murmurs?

Dr. Paul: I should think the course was consistent with severe rheumatic heart disease alone. The murmurs themselves were probably consistent with that diagnosis.

Clinical Diagnoses

Rheumatic heart disease, with mitral stenosis and regurgitation.
Embolism of left middle cerebral artery.

Dr. Paul's Diagnoses

Rheumatic heart disease, with mitral stenosis and regurgitation.
Rheumatic endocarditis, chronic, aortic?
Cardiac hypertrophy and dilatation, especially left auricle.
Mural thrombus, left auricle.
Pulmonary infarcts, healed.
Cerebral embolus.
Subacute bacterial endocarditis?
Chronic passive congestion of spleen.

Anatomical Diagnoses

Rheumatic heart disease, with mitral stenosis and insufficiency.
Mural thrombus, left auricle.
Cerebral embolus, left middle cerebral artery.
Endocarditis, chronic rheumatic, slight, aortic and tricuspid.
Cardiac hypertrophy and dilatation, especially left auricle.
Rheumatic endocarditis, chronic, aortic.
Chronic passive congestion of spleen and liver.
Renal infarcts, healed.

Pathological Discussion

Dr. Tracy B. Mallory: At post-mortem examination the heart was enormously dilated but not very much hypertrophied; it weighed 400 gm., which is perhaps 30 per cent above normal for a woman of this size. The cavity of the various chambers was estimated to contain about 100 cc. on the left and 200 cc. on the right; the right auricle contained 300 cc., and the left auricle 1000 cc. The mitral valve was severely stenotic and also probably showed regurgitation. There were only traces of rheumatic involvement on the aortic and tricuspid valves. I should think it doubtful if either of them was functionally inadequate. The lungs revealed comparatively little evidence of chronic passive congestion. There was a scar of an old pulmonary infarct of considerable size. The kidneys likewise showed scars of old healed infarcts, and there was a fresh embolus in the middle cerebral artery, with softening of the left cerebral hemisphere. The source of the embolus was a large thrombus in the enormously dilated left auricle.

Dr. Richard J. Clark: What was the size of the spleen?

Dr. Mallory: It weighed 275 gm. That is about an average weight for the spleen of chronic passive congestion and rheumatic heart disease. I doubt if it could have been palpated as described in the record. It is rare that a spleen less than 400 gm. can be felt.

Dr. Clark: Was there any positive evidence of active rheumatic infection?

Dr. Mallory: None whatever, grossly or microscopically.

An index of the diagnoses and discussers of the Case Records for the year 1947 will be found elsewhere in this issue of the *Journal*.

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THE JOURNAL does not hold itself responsible for statements made by any contributor.

COMMUNICATIONS should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston 15, Massachusetts.

JANUARY 1, 1948

BEARING the date January 1, 1948, Number 1 of the two hundred and thirty-eighth volume of the *New England Journal of Medicine* will shortly make its New Year bow, one hundred and twenty years — less one month and nineteen days — after the first issue of the *Boston Medical and Surgical Journal* made its own debut.

This does not mean, however, that February 19, 1828, marked the beginning. The *Boston Medical and Surgical Journal* did not on that date spring into being fully edited, printed and proofread, like Athena from the brow of Zeus. Rather did it result from the happy union of two reputable journals of the day — the *New England Journal of Medicine and Surgery and the Collateral Branches of Science*, born in January, 1812, and the *Boston Medical Intelligencer*, which had appeared at weekly intervals with perfect regularity from April 29, 1823, until February 12, 1828.

These historic facts, probably of local interest alone, have occasionally been published in our paper during the past century or so. The present juxtaposition of dates seems to warrant another casual allusion to them. Also, as a right of way must be maintained by the erection of a temporary barrier at established intervals, so must the *Journal* protect its obvious right to the distinction of being the oldest medical periodical in continuous existence by occasional reference to the fact.

It is certainly the direct lineal descendant of the *New England Journal of Medicine and Surgery and the Collateral Branches of Science* that saw daylight one hundred and thirty-five years ago next month, and is thus senior by eleven years even to the venerable and esteemed *London Lancet*.

Like the old (Boston) lady's favorite brand of tea that "had a firm grip on the third water," New England folk and New England institutions often move through their successive generations with a vigor apparently undiminished by the passage of time.

So may it long continue to be with the *New England Journal of Medicine* that now, temporarily suspending its customary restraint, wishes a Happy New Year to its readers, its board, its publishers and its advertisers and particularly to the loyal office personnel that makes its continued appearance not only possible but probable.

PANDORA'S BOX

TWO papers appearing elsewhere in this issue of the *Journal* call particular attention to the sinister role played by the human carrier in the transmission of infectious disease. This is no new idea; further evidence simply adds weight to facts already known or suspected and fastens onto the carrier more firmly his responsibility in this respect.

Lemon, in his medical progress report "The Nasal Carrier of Beta-Hemolytic Streptococci" presents a special indictment of this particular type of public enemy. Nasal carriers, according to studies of environmental contamination made during the War by the Commission on Air-Borne Infection, disseminate at least one hundred times the number of or-

ganisms from the respiratory tract that carriers with pharyngeal implantation only are able to produce.

The real offenders, these studies show, are the patients with clinical sinus disease, "seeding" their environments with as many as eleven million pathogenic micro-organisms at one nose blow! As further evidence of the distinction between nasal and mere pharyngeal carriers, one investigator found that cultures from the hand washings of pharyngeal carriers yielded an average of 4700 streptococci per test, and those from the nasal carriers an average of 790,000. The nasal carrier state, however, tends to be shorter than the more chronic pharyngeal carrier state.

Clifford, discussing epidemic diarrhea of the newborn in a paper read before the Massachusetts Medical Society, in May, 1947, shows that human carelessness, combined with poor and crowded hospital conditions, is the most important factor in the spread of this lethal infection. It is a disease that has followed in the wake of prosperity and the increased demand for hospitalization for obstetric care — a demand with which the available facilities have been unable to keep pace. It is a scourge of the innocent that lends substance to serious consideration of a return to home deliveries and the exclusive hospital isolation of mother and infant together.

A British experience along similar lines was made the subject of recent editorial comment in the *Journal*.[1] In a crowded hospital nursery in England, a staphylococcus giving rise to outbreaks of pemphigus neonatorum and conjunctivitis showed its highest environmental incidence in the noses of the nursing staff.

Rivers,[2] in the Cutter Lecture delivered at the Harvard Medical School in April, 1947, shows, among other things, how dangerously we may contaminate our environment with fecal material. As an example an infant infected with hookworm was kept on a small cotton blanket for twenty-four hours, with no apparent fecal contamination resulting. At the end of five days, however, 20,000 infective hookworm larvae were obtained from the blanket.

And so each of us goes his separate way, each at times sowing the seeds of disease throughout his environment in proportion to his opportunities, his ignorance, his carelessness and his lack of conscience. Most diseases cannot be eradicated and the amelioration of their ravages, as Rivers suggests, may perhaps rest finally in a happy and harmless state of mutual symbiosis between parasite and host. This is a state, however, gradually to be attained with each group of infective organisms. It does not preclude a continuing and increasing vigilance over eating habits, places and utensils; pocket handkerchiefs and their proper use; face towels and toothbrush holders; and doorknobs, faucet handles and all the various objects upon which we so casually deposit our invisible agents of destruction.

So long as these are the facts it behooves us all to maintain a certain degree of physiologic reserve and to practice keeping at least our noses out of other people's business.

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1. Editorial. Epidemiology of neonatal skin infection. *New Eng. J. Med.* 237:528, 1947.
2. Rivers, T. M. Certain public health aspects of infectious disease. *New Eng. J. Med.* (in press).

THE MASSACHUSETTS MEDICAL SOCIETY AND THE BOSTON MEDICAL LIBRARY

Last February the Council of the Massachusetts Medical Society, realizing the great value of the Boston Medical Library to the entire medical profession of Massachusetts, and recognizing its need of increased income if it is to continue its services, graciously agreed to take a substantial part in its support. It agreed to allocate $5.00 from the dues of each member to the Library annually.

In return the Library has created a new type of membership to be known as "Massachusetts Medical Society Members," and has invited the Council to elect four members to the Board of Trustees, and will offer generous privileges to these new members.

This annual contribution will increase the income of the Library by about $25,000. This will allow the Library to carry out its plan to expand its services by increasing its personnel, offering eventual delivery service of books and pamphlets to its fellows and members, increasing its file of periodicals and enlarging and making more available its collection of books.

The fellows of the Library now pay $15.00 a year in dues; with the smaller dues of the various classes of members this constitutes a considerable source of

income. Some fellows may mistakenly believe that with the additional increase from the Massachusetts Medical Society members their dues will no longer be required. The combined income from the Library's investments and dues and the Massachusetts Medical Society members will just about carry the contemplated increase in services, leaving nothing over for the very necessary completion of seven unfinished tiers of stacks, the refurnishing of Holmes, Sprague and Ware halls and the completion of the also unfinished Periodical Room. If money is borrowed for these structural changes (and they are necessary if the Library is to fulfill its mission) it is evident that not only all this income but more will be necessary. It follows, then, not only that all fellows must retain their membership but also that more must be acquired.

The fellows constitute the legal corporation of the Library, control its assets and real estate and elect its officers. When funds are obtained to refurnish parts of the building it is planned to set aside certain rooms for the exclusive use of the fellows. To be a fellow of the Boston Medical Library will continue to be a real distinction.

SUBSCRIPTION RATE INCREASES

The *New England Journal of Medicine*, after twenty-seven years without an increase in subscription rate, finds it necessary at last to pass on to its subscribers part of the growing cost of publication. After January 1, 1948, the regular subscription rate will be $7.00 per annum.

MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH

CONSULTATION CLINICS FOR CRIPPLED CHILDREN IN MASSACHUSETTS UNDER THE PROVISIONS OF THE SOCIAL SECURITY ACT

Clinic	Date	Clinic Consultant
Salem	January 5	Paul W. Hugenberger
Haverhill	January 7	William T. Green
Brockton	January 8	George W. Van Gorder
Lowell	January 9	Albert H. Brewster
Greenfield	January 12	Charles L. Sturdevant
Gardner	January 13	Carter R. Rowe
Worcester	January 16	John W. O'Meara
Fall River	January 19	David S. Grice
Springfield	January 20	Garry deN. Hough, Jr.
Pittsfield	January 21	Frank A. Slowick
Hyannis	January 22	Paul L. Norton

Physicians referring new patients to clinics should get in touch with the district health officer to make appointments. Patients are seen by appointment only.

NOTICES

JOSEPH H. PRATT DIAGNOSTIC HOSPITAL

Bennet Street, Boston
Lecture Hall, 9–10 a.m.

Medical Conference Program

Wednesday, January 7 — Histochemical Studies of Blood and Bone Marrow. Dr. Jack Rheingold.

Friday, January 9 — Carcinoma of the Prostate. Dr. George G. Smith.

Wednesday, January 14 — Pediatric Clinicopathological Conference. Drs. James M. Baty and H. E. MacMahon.

Friday, January 16 — Diagnosis of Thyroid Deficiency and the Use of Thyroid Hormone Therapy in Infants, Children and Adolescents. Dr. Nathan B. Talbot.

Wednesday, January 21 — Recurrent Spontaneous Pneumothoraces and Their Surgical Treatment. Dr. Francis M. Woods.

Friday, January 23 — Rheumatic Fever from the Epidemiological and Preventive Points of View. Dr. David D. Rutstein.

Wednesday, January 28 — The Tuberculoid Structure. Dr. Francis M. Thurmon.

Friday, January 30 — The Clinical Significance of Various Types of Peripheral Neuritis. Dr. D. Denny-Brown.

On Tuesday and Thursday mornings from 9:00 to 10:00, Dr. S. J. Thannhauser will give medical clinics on hospital cases. On the second and fourth Friday afternoons of each month, therapeutic conferences will be held from 2:00 to 4:00 with round-table discussion, Dr. R. P. McCombs, moderator; Dr. Merrill Sosman will conduct x-ray conferences from 4:00 to 6:00. On Saturday mornings from 9:00 to 10:00 clinics will be given by Dr. William Dameshek. Medical rounds are conducted each weekday except Saturday by members of the staff from 12:00 to 1:00.

All exercises are open to the medical profession.

TUFTS ALPHA OMEGA ALPHA

The Tufts chapter of the Alpha Omega Alpha will meet at the Boston Medical Library, 8 Fenway, on Wednesday, January 7, at 8 p.m. Dr. Siegfried J. Thannhauser and Dr. H. Edward MacMahon will conduct a clinicopathological conference.

NEW ENGLAND HOSPITAL FOR WOMEN AND CHILDREN

The monthly clinical conference and meeting of the staff of the New England Hospital for Women and Children will be held on Thursday, January 8, at 7:15 p.m. in the classroom of the Nurses' Residence. The subject, "Subarachnoid Hemorrhage," with a case presentation, will be discussed by Dr. Edith D. Stanley. Dr. Eleanor E. Cowan will be chairman.

Since this is the annual meeting, a full attendance is requested. The change of date from January 1 to January 8 should be noted.

CONFERENCE ON NORMAL AND PATHOLOGIC PHYSIOLOGY OF PREGNANCY

The Committee on Human Reproduction of the National Research Council, in behalf of the National Committee on Maternal Health, will sponsor a conference on the normal and pathologic physiology of pregnancy. The chairman will be Dr. Earl T. Engle, assisted by Dr. Deborah C. Leary, of the National Research Council. Morning and afternoon sessions will be held at the Hotel Pennsylvania on January 30 and 31.

Among those participating in the four sessions are Drs. Arthur T. Hertig, Don Wayne Fawcett, George Van S. Smith and Foster Kellogg and Mrs. Bertha S. Burke.

(*Notices continued on page xi*)

INDEX

TO

The New England Journal of Medicine

Volume 237, July 3, 1947 to December 25, 1947

PAGES ACCORDING TO WEEKLY ISSUES

Pages	No.	Date
1– 38	1	July 3
39– 70	2	July 10
71–110	3	July 17
111–140	4	July 24
141–174	5	July 31
175–206	6	Aug. 7
207–242	7	Aug. 14
243–294	8	Aug. 21
295–348	9	Aug. 28
349–382	10	Sept. 4
383–418	11	Sept. 11
419–464	12	Sept. 18
465–500	13	Sept. 25
501– 530	14	Oct. 2
531– 568	15	Oct. 9
569– 602	16	Oct. 16
603– 650	17	Oct. 23
651– 682	18	Oct. 30
683– 718	19	Nov. 6
719– 754	20	Nov. 13
755– 794	21	Nov. 20
795– 828	22	Nov. 27
829– 886	23	Dec. 4
887– 930	24	Dec. 11
931– 968	25	Dec. 18
969–1004	26	Dec. 25

BOOK REVIEWERS

Abbott, John A.
Allen, Frank N.
Appel, Bernard
Augustine, Donald L.
Ballard, James F.
Barton, Robert B.
Belding, David L.
Benda, Clemens E.
Brodny, M. Leopold
Brown, Ethan A.
Brues, Austin M.
Buck, Robert W.
Castleman, Benjamin
Chute, Richard
Cohen, Mandel E.
Colby, Fletcher H.
Colcock, Bentley P.
Daland, Ernest M.
Dameshek, William
Dearborn, Lester W.
Deutsch, Emmanuel
Dodge, Carroll V.
Downing, John G.
Edwards, Edward A.
Farber, Sidney
Faulkner, James M.
Faxon, Nathaniel W.
Feemster, Roy F.
Fine, Jacob
Finland, Maxwell
Fitz, Reginald
Freedman, Harold J.
Friedman, Emerick
Goldberg, Bernard I.
Goodale, Robert L.
Gould, L. V.
Green, Robert M.
Guthrie, Riley H.
Hayden, E. Parker
Hirsch, Oskar
Hooker, Sanford
Joslin, Elliott P.
Kazanjian, Varaztad H.
Kelemen, George
Kettle, Ronald H.
Krumbhaar, George D.
Kuhns, John G.
Lahey, Frank H.
Lane, C. Guy
Larson, Carroll B.
Leary, Timothy
Leavitt, Peirce H.
Lever, Walter F.
Lippman, Fritz
Lombard, Herbert L.
Lowry, Franklin P.
Lurie, Moses H.
McCarthy, Francis P.
Marble, Alexander
Meigs, Joe V.
Meltzer, Philip E.
Moore, Merrill
Morrison, Hyman
Moses, Sanford H.
Muellner, S. Richard
Myerson, Abraham
O'Brien, Frederic W.
O'Hara, Dwight
Osgood, Robert B.
Phaneuf, Louis E.
Pope, Alton S.
Quigley, Thomas B.
Resnik, Joseph
Robey, William H.
Root, Howard F.
Savitz, Harry A.
Schmidt, H. O.
Segal, Maurice S.
Shattuck, George C.
Smith, George G.
Smith, Richard M.
Sosman, Merrill C.
Spector, Benjamin
Stetson, Charles G.
Stone, Moses J.
Tucker, Arthur S.
Viets, Henry R.
Waite, J. Herbert
Walker, Burnham S.
Warren, Kenneth W.
Wesselhoeft, Conrad
Wheeler, Ralph E.
White, Franklin W.
White, Paul D.
Wiggin, Sidney C.
Wittenberg, H. W.

KEY TO ABBREVIATIONS

c — correspondence
cr — case record
e — editorial
ma — medicolegal abstract
mdph — Massachusetts Department of Public Health
mms — Massachusetts Medical Society
mp — medical progress
misc — miscellany
mr — meeting report
n — notice
ness — New England Surgical Society
o — obituary
* — original article

SUBJECT INDEX

A

ABDOMEN. Wounds [Welch] 156, 187 – mp
ABDUCENS nerve. Paralysis. Complications [Rose & Pritzker] 52*
ABORTION. Induced [Paganini & Wesselhoeft] 173 – c
ABSCESS. Subpectoral [Lawrence & Anglem] 390*
  Subphrenic [Hurwitt] 398*
ADDISON'S DISEASE [Adler] 805*
  [Levenson & others] 152*
ADENOMA [Miller] 198 – cr
AEROSOL [Bryson & Grace] 683*
ALLERGY 64 – e
ALVARENGA prize 600 – misc
AMEBIASIS [Nicholson & De Dominicis] 799*
AMERICAN ACADEMY OF ALLERGY, Dec. 15-17 602 – n
AMERICAN ALLERGY FUND grants-in-aid for scientific research 500 – n
AMERICAN ASSOCIATION ON MENTAL DEFICIENCY, Oct. 18, 568; May 11-15, 1948, 140 – n
AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY, Feb. 6, 1948, 242 – n
AMERICAN COLLEGE OF SURGEONS. Schedule 1948 930 – n
AMERICAN MEDICAL ASSOCIATION 827 – e
  House of Delegates 345 – e
  Questionnaire. Postwar 36 – mms
American Medical Directory 885 – misc
AMERICAN SOCIETY OF ANESTHESIOLOGISTS, Incorporated and New England Society of Anesthesiologists, Nov. 10, 530 – n
AMYLOIDOSIS [Levenson & others] 152*
ANDERSON, DONALD G. 204 – misc
ANEMIA. Acute hemolytic [Colmers & Snavely] 505*
ANESTHESIA [Marcus & others] 258*
  Intravenous [McCann] 931*
  Spinal [Rose & Pritzker] 52*
ANEURYSM [Simeone] 921 – cr
ANNOUNCEMENTS
  Allman, Sydney J. 602
  Blank, Edward 70
  Clauser, William J. 602
  Cohen, Newman 794
  Colloten, Frank B. 529
  Delman, A. H. 602
  Dine, Robert F. 754
  Goldberg, Irving E. 38
  Grace, Sydney 38
  Hoffman, Howard A. 602
  Jacobson, Bernard M. 968
  Kant, Otto 38
  Louis, Nathan 754
  Mason, Robert L. 886
  Massell, Benedict F. 294
  Rouillard, Francis 886
  Sever, James W. 754
  Tait, H. Sinclair 602
  Tate, John C. 38
  Webster, Richard C. 754
  Wiggin, Sidney C. 602
ANTIBIOTIC agents [Long] 837 – mms
ANTIRABIC VACCINE 826 – e
  Reactions 828 – mdph
AORTA. Stenosis [Williams & Bland] 590 – cr; [Sprague & others] 958 – cr
APPOINTMENTS
  American College of Surgeons. Fellows 600 – misc
  Hooper, Francis W. 204
  Lutman, Grace E. 204
  Wellock, William D. 204
  Wood, William W. 204
ARMY MEDICAL CENTER 292 – e
ARTERIES. Roentgenology [Schatzki] 738 – mp
ARTERY. Renal [Goodyear & Beard] 355*
ARTHRITIS. Rheumatoid [Browning & others] 428*
  [Ludwig] 404 – cr
ASCITES. Surgery [Welch] 735*
ASSOCIATION OF MILITARY SURGEONS of United States, Nov. 13-15, 718 – n
ASTHMA [Wiswell & Rackemann] 364, 400 – mp
AUTOMOBILES. Speeding 495 – e

B

BENADRYL [Loveless & Brown] 501*
BIGELOW, HENRY JACOB, Gold Medal. Education of surgeon [Cutler] 466*
  Introductory remarks and presentation [Meigs] 465*
BILIRUBIN. Test [Holt] 580*
BIOPSY [Volwiler & Jones] 651*
BLOOD program 527 – e
BLUE CROSS 138 – e
BLUE SHIELD [Hayden] 901 – mms
BONES. Cyst [Verstandig] 21*
BOOK REVIEWS
  Acetanilid [Gross] 205
  Adolescent Sterility [Montague] 381
  Adrian, E. D. 968
  Agnosia, Apraxia, Aphasia [Nielsen] 173
  Albrecht, F. K. 70
  Allen, R. B. 205
  Allergy in Theory and Practice [Cooke] 417
  American Hospital [Corwin] 174
  Anatomy of Bronchial Tree [Brock] 416
  Anderson, O. W. 379
  Anesthesia in General Practice [Cullen] 752
  Ashburn, P. M. 753
  Baxter, J. P., 3rd 206
  Benda, C. E. 174
  Bierring, W. L. 968
  Biographies of Presidents of American Medical Association [Bierring] 968
  Boas, L. C. 380
  Brock, R. C. 416
  Carbohydrate Metabolism [Soskin & Levine] 173
  Cardiovascular Diseases [Scherf] 417
  Centennial of Surgical Anesthesia [Fulton & Stanton] 205
  Chest [Rigler] 174
  Christian, H. A. 752
  Clinical Methods of Neuro-ophthalmologic Examination [Kestenbaum] 347
  Collens, W. S. 380
  Conduction Anesthesia: Clinical studies of George P. Pitkin 206
  Conel, J. L. 793
  Cooke, R. A. 417
  Corwin, E. H. L. 174
  Cullen, S. C. 752
  Curare [McIntyre] 967
  Denny-Brown, D. 600
  Development of Inhalation Anesthesia [Duncum] 793
  Development of Modern Medicine [Shryock] 886
  Diabetes [John] 380
  Diagnosis and Treatment of Bronchial Asthma [Gay] 205
  Diagnosis and Treatment of Menstrual Disorders and Sterility [Mazer & Israel] 381
  Diseases of Basal Ganglia and Subthalamic Nuclei [Denny-Brown] 600
  Diseases of Chest, with Emphasis on X-ray Diagnosis [Rubin] 967
  Dollar, M. L. 379
  Duncum, B. M. 793
  Eggston, A. A. 968
  Ego and Mechanisms of Defense [Freud] 415
  Elman, R. 416
  Ewerhardt, F. H. 499
  Fidler, A. 464
  Fishbein, M. 968
  Frantz, V. K. 174
  Freud, A. 415
  Fulton, J. F. 205
  Fundamentals of Clinical Neurology [Merritt & others] 567
  Gastroenterology in General Practice [Pelner] 416
  Gay, L. W. 205
  Goni, A. R. 380
  Gross, M. 205
  Handbook of Commonly Used Drugs [Pijoan & Yaeger] 567
  Handbook to Correctional Psychology 886

Harvey, H. D. 174
Hathaway, K. B. 529
Health Insurance in United States [Sinai & others] 379
Herz, E. 70
Histopathology of Ear, Nose and Throat [Eggston & Wolff] 968
History of American Medical Association 1847 to 1947 [Fishbein] 968
Hoch, P. H. 108
Hodges, F. J. 463
Holt, J. F. 463
Hudson, E. H. 415
Hyman, H. T. 416
Integrated Practice of Medicine [Hyman] 416
Introduction to Surgery [Frantz & Harvey] 174
Israel, S. L. 381
John, H. J. 380
Johnson, H. C. 567
Journals and Letters of Little Locksmith [Hathaway] 529
Kalinowsky, L. B. 108
Kestenbaum, A. 347
Lampe, I. 463
Levine, R. 173
Manual of Tomography [Weinbren] 347
Mazer, C. 381
McIntyre, A. R. 967
Medical Education and Changing Order [Allen] 205
Medical Research 206
Merritt, H. H. 567
Mettler, F. A. 567
Military Neuropsychiatry 567
Modern Management in Clinical Medicine [Albrecht] 70
Modern Treatment of Diabetes Mellitus [Collens & Boas] 380
Mongolism and Cretinism [Benda] 174
Montague, M. F. A. 381
Moore, J. E. 463
Motor Disorders in Nervous Diseases [Herz & Putnam] 70
Murphy, D. P. 417
Myasthenia Gravis [Goni] 380
Nielsen, J. M. 173
Parenteral Alimentations in Surgery [Elman] 416
Pelner, L. 416
Penicillin in Neurology [Walker & Johnson] 567
Penicillin in Syphilis [Moore] 463
Personality of Preschool Child [Wolff] 752
Physical Background of Perception [Adrian] 968
Pijoan, M. 567
Postnatal Development of Human Cerebral Cortex [Conel] 793
Principles and Practice of Medicine [Christian] 752
Principles of Surgical Treatment [Rubin] 967
Psychiatric Interviews with Children 380
Psychoanalytic Study of Child 793
Putnam, T. J. 70, 567
Radiology for Medical Students [Hodges & Holt] 463
Ranks of Death [Ashburn] 753
Rhoades, W. 753
Riddle, G. F. 499
Rigler, L. G. 174
Robinson, V. 206, 499
Rubin, E. H. 967
Rubin, M. 967
Scherf, D. 417
Scientists against Time [Baxter] 206
Second Forty Years [Steiglitz] 174
Self You Have to Live With [Rhoades] 753
Shock Treatments and Other Somatic Procedures in Psychiatry [Kalinowsky & others] 108
Shryock, R. H. 886
Sinai, N. 379
Soskin, S. 173
Stanton, M. E. 205
Steiglitz, E. J. 174
Steindler, A. 109
Studies and Essays in the History of Science and Learning Offered in Homage to George Sarton on Occasion of his Sixtieth Birthday 753
Surgical Treatment of Soft Tissues 348
Therapeutic Exercise [Ewerhardt & Riddle] 499
Traumatic Deformities and Disabilities of Upper Extremity [Steindler] 109
Treponematosis [Hudson] 415
Urgent Surgery 381
Uterine Contractibility in Pregnancy [Murphy] 417
Victory over Pain [Robinson] 499
Walker, A. E. 567
Wassersug, J. D. 416
Weinbren, M. 347
White Caps: Story of nursing [Robinson] 206
Whither Medicine: from dogma to science? [Fidler] 464
Wolff, D. 968
Wolff, W. 752
Yaeger, C. H. 567
Your Rheumatism and Backaches [Wassersug] 416
BOSTON COMMUNITY FUND 558 – e
BOSTON MEDICAL LIBRARY 1003 – e
Report of Librarian 14*
Report of President 12*
BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY, Nov. 20, 718 – n
BOSTON UNIVERSITY appointments 414 – misc
BOWERS, WALTER PRENTICE 202 – e; 497 – o
BREAD. Salt-Free [Contratto] 172 – c
BREAST. Carcinoma [Taylor & Wallace] 475*
BRONCHI. Tumors [Miller] 198 – cr
BRONCHIECTASIS [Mallory] 795 – mms
[Badger] 937 – mms
BURNS. Thermal [Levenson & others] 152*
BURRAGE, WALTER S. 929 – misc

C

CANCER 928 – mdph
Cytology test 929 – mdph
CANCER INSTITUTE, Nov. 16-21, 750
CANCER mortality 884 – mdph
Research. Grants and fellowships 294 – n
CARBON tetrachloride [Clinton] 183*
CARCINOMA [Adams] 875 – cr
[Bartlett & Ingersoll] 708 – cr
[Davenport] 408 – cr
[Dunphy] 111*
[Fremont-Smith & others] 302*
[Hamlin & Jefferies] 960 – cr
[Jameson & Mullins] 699 – ness
[Moore] 786 – cr
[Ottenheimer] 1 – ness
[Rose] 878 – cr
[Sanchez-Ubeda & Carr] 87*
[Sarris] 284 – cr
[Simeone] 782 – cr
[Taylor & Wallace] 475*
[VanderLaan] 221*
CELL-BLOCK technic [Chapman & Whalen] 215*
CERVIX. Cicatrix [Meigs] 746 – cr
CHEMOTHERAPY 925 – e
CHICKEN POX [Cohen & Bansmer] 222*
CHILD health service 679 – e
CHILDREN'S HOSPITAL. Lecture July 23, 70 – n
New director 925 – e
CHOLANGITIS. Intrahepatic [Ellis] 372 – cr
CHRISTMAS 963 – e
CHRISTMAS Seals 826 – e
COCCIDIOIDOMYCOSIS [Kurz & Loud] 610*
COLD. Common 202 – e
COLLEGE OF PHYSICIANS fellowships 206 – n
COLON. Tumors [Robbins] 489 – cr
COMMON bile duct. Diseases [Linton & others] 99 – cr
[Stillman] 60 – cr
CONTRACEPTIVE care [O'Brien] 966 – c
to married women [DeNormandie] 682 – c
CORRESPONDENCE. Unauthorized [Kane] 204 – c
COUNCIL. October meeting 648 – e
CUSHING VETERANS ADMINISTRATION HOSPITAL, Oct. 24-31, 602 – n
CUTLER, ELLIOTT CARR 681 – o
CUTTER LECTURE, Nov. 24, 754 – n
CYST. Bronchiogenic [Aub] 743 – cr

D

DEATHS
Alpert, Louis 649
Arkin, Louis 649
Avedisyan, Avedis D. 883

Baum, Ewald G. 791
Baxter, G. Raymond 293
Bean, Charles F. K. 883
Blair, Orrin C. 293
Bowers, Walter P. 202 – e; 497 – o
Brown, Martin M. 560
Charron, Ovide T. 560
Clarke, Joshua W. 66
Cowles, Frank A. 560
Coyne, Thomas J. 964
Cutler, Elliott C. 378; 681 – o
Davis, Max 172
Delano, Samuel 883
Dolloff, Charles H. 649
Donovan, Thomas R. 66
Dutton, Frank K. 172
Flynn, Timothy P. 649
Gilman, Florence 293
Goodall, Edwin B. 293
Gorham, George H. 172
Grady, Henry M. 240
Harris, Charles E. 240
Hawkins, Henry 598
Henderson, Francis F. 883
Hennessey, Thomas F. 240
Higgins, George V. 560
Howard, Allen R. 107
Howe, W. Lewis 378
Johnson, Mary W. L. 560
Kelly, William P. 107
King, Frederick A. 791
Kinloch, Raymond A. 204
Lucas, Julian D. 598
Menard, Leon J. 964
Morrill, Herman A. 139
Murphy, Harold A. 598
Nelson, Christian A. 680
Nightingale, James 293
Nye, Robert N. 493 – e; 751 – o
O'Hara, Francis J. 107
Potter, Lester F. 528
Preble, Wallace 204
Roy, Joseph N. 293
Safford, M. Victor 649
Sanborn, Mary N. 649
Shatswell, James A. 528
Sibley, Benjamin E. 883
Sikorsky, Vladimir N. 463
Spiegl, Erwin D. 528
Stone, Jane Gray 964
Underhill, Elizabeth C. 680
Wentworth, Arthur H. 293
Wise, John M. 378
DERMATOSES. Occupational [Downing] 755 – mms
DIABETES mellitus [Adler] 805*
DIAGNOSIS [Thompson & Rogers] 965 – c
DIARRHEA. Newborn [Clifford] 969 – mms

E

EATON, MONROE DAVIS 204 – misc
ECZEMA vaccinatum [Rubenstein] 395*
EDITOR. New 679 – e
EDITORIALS
Allergy to influenza A and B vaccines 64
Antirabic vaccine 826
Army medical center 292
Bewildered age 65
Bowers, Walter Prentice 202
Chemotherapy of tuberculosis 925
Child health service for student veterans 679
Children's Hospital. New director 925
Christmas 963
Christmas seal campaign 826
Common cold. Research 202
Council. October meeting 648 – mms
De Senectute 36
Editor, new. Appointment 679
Exudative tonsillitis and pharyngitis of unknown etiology 170
Fitting memorial (Nye) 598
Folic acid. Warning 713
Greater Boston Community Fund 558
Greater Boston Nursing Council 413
Health centers 377
Heart disease. Trend 139
Hospital care. Cost 461
Hospital services 138
House of Delegates (A.M.A.) 345
Hypertension. Dietary treatment 748
Influenza 881
Isolation 963
Lead poisoning. Epidemic 714
Madame president — American Public Health Association (Eliot, M. M.) 749
Massachusetts Medical Society and Boston Medical Library 1003
Medical group practice 106
Medical writing. Fashions 376
"Mercy cars" 495
New England Postgraduate Assembly 596, 882
New Year 1002
Nontuberculous pulmonary calcification 171
Nye, Robert Nason 493
Pandora's box 1002
Passing era in medical history 106
Pediatrics. Survey 238
Pleurodynia. Epidemic 649
Poliomyelitis. Epidemiology 462
Q fever in United States 290
Quantity versus quality 596
Red Cross national blood program 527
Rubella syndrome. Study 791
Skin. Infection. Neonatal 528
Streptococcal infections. Sulfadiazine-resistant 558
Streptomycin in bacterial endocarditis 680
Streptomycin. Toxicity 493
Subacute and malignant form of epidemic hepatitis 35
Subscription rate increases 1004
Tetanus 411
Thanksgiving 790
Toxoplasmosis 346
Veterans. Medical care 237
Woman's auxiliaries 827
ELECTROENCEPHALOGRAMS [Kibbe] 122*
ELIOT, MARTHA MAY 749 – e
EMBOLISM [Giddings] 135 – cr
EMERSON, KENDALL. Resignation 752 — misc
ENDOCARDITIS [Ross and others] 32 – cr
Bacterial 680 – e; [Paul & others] 349*
ENDOSCOPY [Benedict] 92, 126 – mp
ESOPHAGEAL [Bryan] 941*
ESOPHAGUS. Stricture [Bryan] 941*
Ulcer [Benedict] 919 – cr
EWING tumor [Franseen] 523 – cr

F

FLUIDS. Serous. Examination [Chapman & Whalen] 215*
FOLIC ACID 713 – e
[Sargent] 667, 703 – mp
FRIEDLANDER bacillus [Solomon] 149*
FROSTBITE [Lange & others] 383*

G

GALL BLADDER. Carcinoma [Sarris] 284 – cr
GALLUPE, H. QUIMBY. Appointment 715 – mms
GASTRECTOMY [MacDonald & others] 887*
Total [Smithwick] 39 ness
GASTRITIS. Emphysematous [Welch & Jones] 983*
GLOMERULONEPHRITIS [Baker] 822 – cr
GOITER. Amyloid [Kranes] 57 – cr
GOLD therapy [Browning & others] 428*
GRANULOMA inguinale [Berkowitz] 665*
GREATER BOSTON MEDICAL SOCIETY, Nov. 18, 754; Dec. 16, 886 – n
GREATER BOSTON NURSING COUNCIL 413 – e
GREENSTEIN, SAMUEL (Restoration of license) [Gallupe] 966 – c
GROSS, ROBERT E. (Matas medal) 752 – misc
GROUP PRACTICE 106 – e
[Hunt] 71*
[Hunt & Goldstein] 719*

GYNECOLOGY [Scheffey] 250*
Anesthesia [McCann] 931*
[Ulfelder & Meigs] 54 – mp

H

HARVARD MEDICAL SCHOOL. Appointments 964 – misc
HARVARD MEDICAL SOCIETY, Oct. 14, 530 – n
HARVEY, SAMUEL C. 929 – misc
HEALTH centers 377 – e
HEART [Ropes] 163 – cr
Abscess [Short] 552 – cr
Disease 139 – e
Fibrosis [Reyersbach] 593 – cr
Rheumatic [Paul] 997 – cr
Roentgenology [Schatzki] 738 — mp
HEMATOLOGY [Dameshek] 434, 480 — mp
HEMATOMETRIUM [Ingersoll] 525 – cr
HEMOGLOBINEMIA [Landsteiner & Finch] 310*
HEMOLYSIS. Intravascular [Burwell & others] 657*
HEPATITIS [Bragdon] 765*
[Young] 225, 261 – mp
Epidemic 35 – e
Infectious [Jersild] 8*
Infectious complications [Martin & Ferguson] 114*
Infectious [Richardson] 369 – cr
HEPATOMA [Clark] 673 – cr
HOSPITAL care 461 – e
HOSPITALS 791 – mdph
and labor 293 – ma
Children's Hospital, lecture, July 23, 70 – n
Cushing Veterans Administration Hospital, Oct. 24-31, 602 – n
Massachusetts Memorial Hospitals. Seizure clinic 500 – n
Metropolitan State Hospital. Postgraduate Seminar 348 – n
New England Hospital for Women and Children, Sept. 4, 294; Nov. 6, 650; Jan. 8, 1004 – n
(Joseph H.) Pratt Diagnostic Hospital, medical conference program, Oct. 8–31, 529; Nov. 14–28, 754; Dec. 10–19, 866; Jan. 7–30, 1004 – n
HOT PACK MACHINES 599 – mdph
HOWARD, H. H. 379 – misc
HUNDRED YEARS AGO 140, 496, 598, 716, 927
HYPERINSULINISM [Sanchez-Ubeda & Carr] 87*
HYPERTENSION [Long & Hinton] 840*
Diet 748 – e
HYPERTHERMIA
[Bragdon] 765*

I

ILEUM. Carcinoma [Bartlett & Ingersoll] 708 – cr
INDUSTRIAL medicine [Tabershaw] 313 – mp
INFECTION. Bacterial [Kane & Foley] 531*
INFLUENZA 881 – e
[Dingle] 845 – mp
Vaccines 64 – e
INTERNSHIP. Liability 883 – ma
INTESTINE. Large. Carcinoma [Dunphy] 111*
INTESTINES. Infection [Pulaski & Amspacher] 419*
Large. Carcinoma [Jameson & Mullins] 699 – ness
Volvulus [Young & others] 78 – ness
INSTITUTE OF HISTORY OF MEDICINE [Rushmore] 498 – e
IRRADIATION [Brick] 48*
[Palmer & Brick] 566 – e
ISOLATION 963 – e

J

JAUNDICE [Heringman & Phillips] 471*
[Young] 225, 261 – mp
Serum 884 – mdph

K

KIDNEY. Carcinoma [Simeone] 782 – cr
Sclerosis [Clark] 819 – cr
KIDNEYS. Injury [Clinton] 183*
KUPPER, MORRIS J. (Restoration of license) 929 – c

L

LABORATORY specimens 108 – mdph
LANGERHANS. Islets. Carcinoma [Sanchez-Ubeda & Carr] 87*
LEAD poisoning. Epidemic 714 – e
LEGISLATIVE CONFERENCE, Oct. 29, 602 – n
LEIOMYOMA [Robbins] 489 – cr
LICENSE. Restoration (Samuel Greenstein) [Gallupe] 966 – c
Restoration (Morris J. Kupper) [Gallupe] 929 – c
LINDEMANN, ERICH 717 – misc
LIPIDS. Serum [Thannhauser] 515, 546 – mp
LIVER. Biopsy [Volwiler & Jones] 651*
Cirrhosis [Brailey] 643 – cr
[Dole] 341 – cr
[Jacobson & others] 231 – cr
LOBOTOMY [Myerson & Myerson] 511*
LUNG. Carcinoma [Schier] 645 – cr
LUNGS. Calcification 171 – e
Carcinoma [Davenport] 408 – cr
Emboli [Clark & others] 132 – cr
LYMPHOMA [Townsend & others] 102 – cr

M

MALPRACTICE. Liability 240 – ma
MANNING, MIRIAM (prize subscription) 752 – misc
MARKLE, JOHN AND MARY, Foundation grants 650 – n
MASSACHUSETTS CENTRAL HEALTH COUNCIL, May 1, 38 – mr; 965 – misc
MASSACHUSETTS DEPARTMENT OF PUBLIC HEALTH
Antirabic vaccine. Reactions 828
Appointments 204
Cancer 928
Cancer. Cytology test 929
Cancer death rate 884
Cancer Institute, Nov. 16–21, 750
Crippled children clinics, July 7–28, 38; Sept. 5–25, 347; Oct. 6–27, 529; Nov. 3–24, 682; Jan. 5–22, 1004
Diseases, communicable, for May, 37; June, 241; July, 414; Aug., 717; Sept., 792; Oct., 885
Elective tonsillectomies during poliomyelitis season 240
Hospital survey and construction, Nov. 12, 650 – n
Jaundice. Serum 884
Laboratory specimens 108
Massachusetts state plan 791
Maternity care 108
Poliomyelitis and tonsillectomy 69
Public-health conference, Sept.11–12, 378
Repatriation of World War II deceased 750
Respirators and hot pack machines 599
Services to physicians [Getting] 693 – mms
Toxin-antitoxin distribution. Discontinued 37
Undulant fever 565
Wassermann Laboratory 204
MASSACHUSETTS hospital facilities 791 – mdph
MASSACHUSETTS MEDICAL SOCIETY
A.M.A. Analysis of postwar questionnaire 36
Antibiotic agents [Long] 837
Applicants for fellowship 560, 598
Boston Medical Library 1003
Bronchiectasis [Badger] 937
Pathogenesis [Mallory] 795
Committee to Confer with General Hawley 238
Council proceedings, May 19, 269; Oct. 1, 853
Council. October meeting 648 – e
Dermatoses. Occupational [Downing] 755
Diarrhea. Newborn [Clifford] 969
Gallupe, H. Quimby, Secretary *pro tempore* 715
Medical care [Hunt] 986
Medical care. Blue Shield [Hayden] 901
Medical care for old-age assistance 172
One hundred and sixty-sixth anniversary, May 20–22, 320
Rathbun, Stenley. Fraud 239
Rehabilitation [Aitken] 903
Services offered to physicians bp Massachusetts Department of Public Health [Getting] 693
Virus diseases [Enders] 897
MASSACHUSETTS MEMORIAL HOSPITALS. Seizure clinic 500 – n
MASSACHUSETTS TUBERCULOSIS LEAGUE. Resolution.
Nye, R. N. 650 – misc
MATAS medal 752 – misc

Maternity care 108 – mdph
Meckel's diverticulum [Moses] 118*
Medical care 596 – e
[Hayden] 901 – mms
Journals wanted [Kazanjian] 108 – c
History 106 – e
[Hunt] 986 – mms
Officers. Increased pay 530 – n
Medicolegal Abstracts
Contempt of court 66
Hospitals and labor 293
Hospitals — Whose servant is intern? 883
Liability for malpractice 240
Medicolegal Conference and Seminar Oct. 13–18, 242 – n
Meningioma. Spinal cord [Watkins] 676 – cr
Meningitis [Solomon] 149*
Mesenteric-artery [Giddings] 135 – cr
Metropolitan State Hospital. Postgraduate Seminar 348 – n
Middlesex South District Medical Society, Oct. 22, 602 – n
Milkman's syndrome [Brick & Bunch] 359*
Mononucleosis. Infectious [Ashworth & Motto] 544*
Moore, Merrill. Award 885 – misc
Motion Pictures. Medical [Leonard] 540*
Muscular dystrophy [Ropes] 163 – cr
Myocarditis [Rogers & Robbins] 829*

N

Nasal carriers 1002 – e
[Lemon] 988 – mp
National Physicians Committee [Frothingham] 792 – c
Nephrosclerosis [Clark] 819 – cr
Neurofibrosarcoma [Hamlin] 234 – cr
Neurology [Merritt & Jordan] 623 – mp
New England Dermatological Society, Oct. 8, 530; Dec. 3, 828 – n
New England Diabetes Association, April 16, 140 – misc
New England Epilepsy League 650 – n
New England Heart Association Dec. 1, 754 – n
Myocarditis [Rogers & Robbins] 829
New England Hospital for Women and Children, Sept. 4, 294; Nov. 6, 650; Dec. 4, 828; Jan. 8, 1004 – n
New England Postgraduate Assembly 596 – e; Oct. 29–31, 882 – e
Gynecology in community [Scheffey] 250
New England Section of the American Urological Association
Transurethral prostatic resection [Nesbit] 207
New England Society of Anesthesiologists, Sept. 16, 348 – n
and American Society of Anesthesiologists, Incorporated, Nov. 10, 530 – n
New England Society of Physical Medicine, Dec. 17, 930 – n
New England State Medical Societies. Council 827
New England Surgical Society
Cancer of rectum [Ottenheimer] 1
Carcinoma of colon and rectum [Jameson & Mullins] 699
Total gastrectomy [Smithwick] 39
Volvulus of cecum and ascending colon [Young & others] 78
New Hampshire Medical Society
Appointments 649
Deaths
Dolloff, Charles H. 649
Flynn, Timothy P. 649
Sanborn, Mary N. 649
Sikorsky, Vladimir N. 463
Proceedings of the One Hundred and Fifty-Sixth Anniversary June 18–20, 442, 630
New Year 1002 – e
New York Academy of Medicine, Oct. 6–17, 348 – n
New York Academy of Sciences, Oct. 3–4, 348 – n
Norfolk District Medical Society. Schedule 242; Sept. 23, 382; Oct. 28, 602; Nov. 25, 794 – n
Norfolk Medical News 600 – misc
Norwood Hospital 929 – misc
Nye, Robert Nason, 493 – e; 751 – o
Memorial 598 – e; 750

O

Obstetrics. Anesthesia [Marcus & others] 258*
Old Age 36 – e
Assistance 172 – mms
Ophthalmology. Research fellowships 965 – misc
Optic atrophy. Syphilitic [Levin & others] 769*
Os calcis [Verstandig] 21*
Ovary. Inflammation [Welch] 995 – cr

P

Pain responses. Cutaneous [Ingraham & others] 773*
Paint hazard [Eley] 929 – c
Pancreas. Carcinoma [Adams] 875 – cr
[Moore] 786 – cr
cyst [Kranes] 194 – cr
Pandora's Box 1002 – e
Pediatrics. Survey 238 – e
Penicillin. Therapy [Goerner & others] 576*
Periarteritis nodosa [Clark] 457 – cr
Pericarditis [Pickering] 486 – cr
Peritonitis. Tuberculous [Brailey] 643 – cr
Phi Delta Epsilon Lecture, Jan. 14, 968 – n
Plasma. Pooled [Hurwitz] 204 – c
Pleurodynia 649 – e
[Finn] 621*
Pneumonia [Scannell & others] 287 – cr
Pneumoperitoneum [Chamberlin] 843*
Poliomyelitis 69, 240 – mdph; 462 – e
Polyneuritis. Infectious [Parker & Adams] 976*
Postgraduate education [Lyman] 600 – c
Pratt, Joseph H., Diagnostic Hospital, medical conference program, Oct. 8–31, 529; Nov. 14–28, 754; Dec. 10–19, 886; Jan. 7–30, 1004 – n
Pregnancy and infectious hepatitis [Martin & Ferguson] 114*
Pregnancy. Conference on normal and pathologic physiology Jan. 30, 31, 1004 – n
Prize subscription 752 – misc
Progress, Medical
Asthma. Chemical factors [Wiswell & Rackemann] 364, 400
Endoscopy [Benedict] 92, 126
Folic acid [Sargent] 667, 703
Gynecology [Ulfelder & Meigs] 54
Hematology [Dameshek] 434, 480
Industrial medicine [Tabershaw] 313
Influenza [Dingle] 845
Jaundice. Current concepts [Young] 225, 261
Lipids. Serum [Thannhauser] 515, 546
Nasal carrier [Lemon] 988
Neurology [Merritt & Jordan] 623
Roentgenology. Diagnostic [Schatzki] 738
Skin. Diseases [Downing] 906, 945
Tuberculosis [Wassersug] 22
Venous pressure [Hussey & Jeghers] 776, 812
Virus infections. Diagnosis [Cheever] 584
War wounds of abdomen [Welch] 156, 187
Prostate. Carcinoma [Rose] 878 – cr
Resection. Transurethral [Landsteiner & Finch] 310*
[Nesbit] 207*
Psychotherapy [Fox] 616*
Pteroylglutamic acid [Sargent] 667, 703 – mp
Purpura [Cohen & Bansmer] 222*
Putnam, Helen, Fellowship 886 – n
Pyelonephritis [Zeller] 454 – cr
Pyribenzamine [Loveless & Brown] 501*

Q

Q Fever 290 – e
[Finland & Lesses] 255*

R

Radio broadcasts 379 – misc
Radiological Society of North America, Nov. 30–Dec. 5, 754 – n
Reading disability [Eustis] 243*
Rectum. Carcinoma [Ottenheimer] 1 – ness
[Schier] 645 – cr
Red Cross 527 – e

Rehabilitation [Aitken] 903 – mms
[Dallas] 943*
Respirators 599 – mdph
Respiratory disease. Therapy [Bryson & Grace] 683*
Rheumatic Fever [Goerner & others] 576*
Research 293 – misc
Rheumatism. Therapy [York & Fischer] 477*
Roche–Organon awards 965 – misc
Roentgenology. Diagnosis [Schatzki] 738 — mp
Rubella 791 – e
Vaccination [Hilliard & Wesselhoeft] 414 – c

S

Salmonella enteritidis [Baker & Bragdon] 175*
Sarcoidosis [Jeffries] 555 – cr
Sarcoma [Hamlin] 234 – cr
Scientific exhibits. Annual meeting [Meigs & Bailey] 379 – c
Septicemia [Baker & Bragdon] 175*
Sickness benefits 108 – misc
Sinus. Pilonidal [Hurwitt] 398*
Skin. Diseases [Downing] 906, 945 – mp
Infection. Neonatal 528 – e
Smallpox. Epidemic [Agerty] 305*
Smears. Vaginal [Fremont-Smith & others] 302*
Societies
American Academy of Allergy, Dec. 15–17, 602 – n
American Association on Mental Deficiency, Oct. 18, 568; May 11–15, 1948, 140
American Society of Anesthesiologists, Incorporated, Nov. 10, 530 – n
Association of Military Surgeons of United States, Nov. 13–15, 718 – n
Boston Society of Psychiatry and Neurology, Nov. 20, 718 – n
Greater Boston Medical Society, Nov. 18, 754; Dec. 16, 886 – n
Harvard Medical Society, Oct. 14, 530 – n
Massachusetts Central Health Council, May 1, 38 – mr
Middlesex South District Medical Society, Oct. 22, 602 – n
New England Dermatological Society, Oct. 8, 530; Dec. 3, 828 – n
New England Diabetes Association, April 16, 140 – misc
New England Heart Association, Dec. 1, 754 – n
New England Society of Anesthesiologists, Sept. 16, 348; Nov. 10, 530 – n
New England Society of Physical Medicine, Dec. 17, 930 – n
Norfolk District Medical Society. Schedule 242; Sept. 23, 382; Oct. 28, 602; Nov. 25, 794 – n
Radiological Society of North America, Nov. 30–Dec. 5, 754 – n
South End Medical Club, Oct. 21, 602; Nov. 18, 754 – n
Suffolk District Medical Society, Dec. 4, 294 (censors' meeting) Dec. 9, 754; Dec. 11, 794 – n
Suffolk District Woman's Auxiliary, Dec. 4, 794 – n
Tufts Alpha Omega Alpha, Jan. 7, 1004 – n
South End Medical Club, Oct. 21, 602; Nov. 18, 754 – n
Spinal cord. Tumors [Watkins & Ballantine] 676 – cr
Spleen. Cyst [Duby] 731*
Stomach. Acidity [Brick] 48*
[Palmer & Brick] 566 – c
Ulcer [Donaldson & others] 167 – cr
Ulcers [Schatzki] 339 – cr
Stools. Culture [Fischer] 53*
Streeter, Edward Clark 106 – e
Streptococcal infections 558 – e
Streptococci [Foley] 809*
Beta-hemolytic [Goerner & others] 576*
[Lemon] 988 – mp
Streptomycin 493, 680 – e
[Kane & Foley] 531*
[Pulaski & Amspacher] 419*
Subscription rate 1004 – e
Suffolk District Medical Society. Censors' meeting, Dec. 4, 294; Dec. 9, 754; Dec. 11, 794 – n
Suffolk District Woman's Auxiliary, Dec. 4, 794 – n
Sulfadiazine 558 – e
Sulfonamides. Hazards [Bryan] 941*
Surgeon. Education [Cutler] 466*
Sympathectomy [Lord & Hinton] 840*

T

Telangiectasia. Familial Hemorrhagic [Kennedy] 180*
Testicles. Tumors [Meltzer & Bloom] 513*
Tetanus 411 – e
Thanksgiving 790 – e
Thrombosis. Renal-Artery [Goodyear & Beard] 355*
Thyroid. Carcinoma [VanderLaan] 221*; [Hamlin & Jefferies] 960 – cr
Tumors [Rogers & others] 569*
Tonsillectomy 69, 240 – mdph
Tonsillitis. Exudative 170 – e
Toxin-antitoxin discontinued 37 – mdph
Toxoplasmosis 346 – e
Tubercle bacilli. Detection [Levinson & White] 186*
Tuberculosis 925 – e
[Ditmore] 702*
[Pickering] 486 – cr
Pulmonary [Chapman] 709 – cr
Rest [Powers] 966 – c
[Wassersug] 22 – mp
Tufts Alpha Omega Alpha, Dec. 10, 886 – n; Jan. 7, 1004 – n
Tufts Medical Journal 752 – misc
Tumor. Ewing [Franseen] 523 – cr
Tumors. Malignant [Rogers & others] 569*

U

Ulcer. Decubitus [Croce & Beakes] 141*
Peptic [Volwiler] 29 – cr
[Benedict] 919 – cr
Undulant fever 565 – mdph
Uremia. Acute. Treatment [Buckley & Scholten] 431*
Urology award 650 – n
Uterus. Carcinoma. Diagnosis [Fremont-Smith & others] 302*

V

Vagotomy [Moses] 603*
Van Meter Prize award 418 – n
Vasospasm [Foisie] 295*
Venous pressure [Hussey & Jeghers] 776, 812 – mp
Veterans 679 – e
Medical care 237 – e; 238 – mms
Virus diseases [Enders] 897 – mms
Infections. Diagnosis [Cheever] 584 – mp

W

War Dead. Returned 750 – mdph
Surgery [Welch] 156, 187 – mp
Wassermann Laboratory 204 – mdph
Waugh, William H. (prize subscription) 752 – misc
Weil's disease [Heringman & Phillips] 471*
Woman's auxiliaries 827 – e
World Health Organization 965 – misc
Wounds. Infections [Solomon] 149*
Writing. Medical 376 – e

Y

Youth Guidance 65 – e

AUTHOR INDEX

A

ADAMS, F. D. 875 – cr
ADAMS, R. D. 976*
ADLER, D. K. 805*
AGERTY, H. A. 305*
AITKEN, A. P. 903 – mms
AMSPACHER, W. H. 419*
ANGLEM, T. J. 390*
ASHWORTH, J. 544*
ASPER, S. P., Jr. 569*
AUB, J. C. 743 – cr

B

BADGER, T. L. 937 – mms
BAILEY, G. G. 379 – c
BAKER, L. M. 428*
BAKER, M. P. 175*; 882 – cr
BALLANTINE, H. T., JR. 676 – cr
BANSMER, C. 222*
BARTLETT, M. K. 708 – cr
BEAKES, C. H. C. 141*
BEARD, D. E. 355*
BELDING, H. W. 887*
BENEDICT, E. B. 92, 126 – mp; 919 – cr
BERKOWITZ, J. 665*
BLAND, E. F. 349*; 590 – cr
BLOOM, B. 513*
BOYD, L. J. 383*
BRAGDON, J. H. 175, 765*
BRAILEY, A. G. 643 – cr
BRICK, I. B. 48, 359*; 566 – c
BROWN, H. 501*
BROWNING, J. S. 428*
BRYAN, B. D. 941*
BRYSON, V. 683*
BUCKLEY, R. W. 431*
BUNCH, R. F. 359*
BURWELL, E. L. 657*

C

CARR, E. A., JR. 87*
CHAMBERLIN, D. T. 843*
CHAPMAN, C. B. 215*
CHAPMAN, E. M. 709, 958 – cr
CHEEVER, F. S. 584 – mp
CLARK, R. J. 132, 819 – cr
CLARK, W. 457, 673 – cr
CLIFFORD, S. H. 969 – mms
CLINTON, M., JR. 183*
COHEN, J. 222*
COLMERS, R. A. 505*
CONTRATTO, A. W. 172 — c
CROCE, E. J. 141*
CUTLER, E. C. 466*

D

DALLAS, H. A. 943*
DAMESHEK, W. 434, 480 – mp
DAVENPORT, L. F. 408 – cr
DE DOMINICIS, F. A. 799*
DENORMANDIE, R. L. 682 – c
DINGLE, J. H. 845 – mp
DITMORE, D. C. 702*
DOLE, V. P. 341 – cr
DONALDSON, G. A. 167 – cr
DOWNING, J. G. 755 – mms; 906, 945 – mp
DUBY, H. 731*
DUNPHY, J. E. 111*

E

ELEY, R. C. 929 – c
ELLIS, D. S. 372 – cr
ENDERS, J. F. 897 – mms
EUSTIS, R. S. 243*

F

FAXON, H. H. 132 – cr
FERGUSON, F. C., JR. 114*
FINCH, C. A. 310, 657*
FINLAND, M. 255*
FINN, J. J., JR. 621*
FISCHER, W. J. H., JR. 53, 477*
FOISIE, P. S. 295*
FOLEY, G. E. 531, 809*
FOX, H. M. 616*
FRANSEEN, C. C. 523 – cr
FREMONT-SMITH, M. 302*
FROTHINGHAM, C. 792 – c

G

GALLUPE, H. Q. 929, 966 – c
GETTING, V. A. 693 – mms
GIDDINGS, W. P. 135 – cr
GOERNER, J. R. 576*
GOLDSTEIN, M. 719*
GOODPASTOR, W. L. 152*
GOODYEAR, W. E. 355*
GRACE, E. J. 683*
GRAHAM, R. M. 302*
GREEN, R. W. 152*
GREENBLATT, M. 769*

H

HAMLIN, E., 234, 960 – cr
HAYDEN, C. G. 901 – mms
HERINGMAN, E. C. 471*
HILLIARD, C. M. 414 – c
HINTON, J. W. 840*
HOLT, C. L. 580*
HUNT, G. H. 71, 719*
HUNT, J. 986 – mms
HURWITT, E. S. 398*
HURWITZ, A. 204 – c
HUSSEY, H. H. 776, 812 – mp]

I

INGELFINGER, F. J. 887*;
INGERSOLL, F. M. 525, 708 – cr
INGRAHAM, F. D. 773*

J

JACOBSON, B. M. 231 – cr
JAMESON, J. W. 699 – ness
JEFFERIES, W. M. 555, 960 – cr
JEGHERS, H. 776, 812 – mp
JERSILD, M. 8*
JONES, C. M. 651, 983*
JONES, T. D. 576*
JORDAN, W. K. 623 – mp

K

KANE, L. W. 204 – c; 531*
KAZANJIAN, MRS. V. H. 108 – c
KENNEDY, H. P. 180*
KIBBE, M. H. 122*
KINNEY, T. D. 657*
KRANES, A. 57, 194 – cr
KURZ, E. R. H. 610*

L

LANDSTEINER, E. K. 310*
LANGE, K. 383*
LAWRENCE, K. B. 390*
LEE, W. V. 428*
LEMON, H. M. 988 – mp
LEONARD, D. W. 540*
LESSES, M. F. 255*2
LEVENSON, S. M. 15*
LEVIN, S. 769*
LEVINSON, L. 186*
LINTON, R. R. 99 – cr
LONG, P. H. 837 – mms
LORD, J. W., JR. 840*
LOUD, N. W. 610*
LOVELESS, M.H. 501*
LUDWIG, A. O. 404 – cr
LUND, C. C. 152*
LYMAN, J. R. 600 – c

M

MACDONALD, R. M. 887*
MALLORY, T. B. 795 – mms
MARCUS, P. S. 258*
MARTIN, R. 114*
MASSELL, B. F. 576*
MATSON, D. D. 773*
MCCANN, J. C. 931*
MEIGS, J. V. 54 – mp; 302*; 379 – c; 465*, 746 – cr
MELTZER, A. 513*
MERRITT, H. H. 623 – mp
MILLER, C. B. 198 – cr
MIXTER, C. G., JR. 99 – cr
MOORE, F. D. 786 – cr
MORRISON, H. R. 78*
MOSES, W. R. 118, 603*
MOTTO, S. A. 544*
MULLINS, C. R. 699 – ness
MYERSON, A. 511*
MYERSON, P. G. 511*

N

NESBIT, R. M. 207*
NICHOLSON, J. H. 799*

O

O'BRIEN, F. W. 966 – c
OTTENHEIMER, E. J. 1 – ness

P

PAGANINI, A. E. 173 – c
PALMER, W. L. 566 – c
PARKER, F., JR. 976*
PAUL, O. 349*; 997 – cr
PHILLIPS, J. H. 471*
PHIPPEN, W. G. 12*
PICKERING, G. W. 486 – cr
POWERS, B. R. 966 – c
PRITZKER, S. 52*
PULASKI, E. J. 419*

R

RACKEMANN, F. M. 364, 400 – mp
REYERSBACH, G. E. 593 – cr
RICE, R. M. 428*
RICHARDSON, W. 102, 369 – cr
ROBBINS, L. L. 489 – cr
ROBBINS, S. L. 829*
ROGERS, J. 829*
ROGERS, W. F., JR. 569*; 965 – c
ROPES, M. 163 – cr
ROSE, A. 878 – cr
ROSE, A. T. 52*
ROSS, R. A. 32 – cr
RUBENSTEIN, A. D. 395*
RUSHMORE, S. 498 – c

S

SANCHEZ-UBEDA, R. 87*
SARGENT, F. II, 667, 703 – mp

SARRIS, P. 284 – cr
SCANNELL, J. G. 287 – cr
SCHATZKI, R. 339, 738 — mp
SCHEFFEY, L. C. 250*
SCHIER, W. W. 645 – cr
SCHOLTEN, R. A. 431*
SCHULZ, M.D. 287 – cr
SHORT, C. L. 552 – cr
SIMEONE, F. A. 782, 921 – cr
SMITHWICK, R. H. 39 – ness
SNAVELY, J. G. 505*
SOLOMON, S. 149*
SPRAGUE, H. B. 958 – cr
STILLMAN, J. S. 60 – cr

T

TABERSHAW, I. R. 313 – mp
TAGNON, H. J. 152*
TAYLOR, F. H. L. 152*
TAYLOR, G. W. 475*
THANNHAUSER, S. J. 515, 546 – mp
THOMPSON, M. 965 – c
TOWNSEND, J. B. 102 – cr
TREVETT, L. D. 769*
TUNNELL, J. W. 258*

U

ULFELDER, H. 54 – mp

V

VANDERLAAN, W. P. 221*
VERSTANDIG, C. C. 21*
VIETS, H. R. 14*
VOLWILER, W. 29, 231 – cr; 651*

W

WALLACE, R. H. 475*
WASSERSUG, J. D. 22 – mp
WATKINS, A. L. 676 – cr
WEINER, D. 383*
WELCH, C. E. 156, 187 – mp; 983*, 995 – cr
WELCH, K. J. 735*
WESSELHOEFT, C. 173, 414 – c
WHALEN, E. J. 215*
WHITE, P. D. 32 – cr; 349*
WHITE, R. L. 186*
WILKINSON, L. H. 258*
WILLIAMS, C. 590 – cr
WILLIAMS, R. H. 569*
WILSON, W. E., Jr. 78*
WISWELL, J. G. 364, 400 – mp
WOODS, R. P. 773*
WYMAN, S. M. 167 – cr

Y

YORK, C. L. 477*
YOUNG, E. L. 78*
YOUNG, L. E. 225, 261 – mp

Z

ZELLER, J. W. 454 – cr

CASE RECORDS
OF THE MASSACHUSETTS GENERAL HOSPITAL

WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*
BENJAMIN CASTLEMAN, M.D., *Associate Editor*
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INDEX

CASE RECORDS OF THE

MASSACHUSETTS GENERAL HOSPITAL

1947

The date of publication of any case may be determined from the case number. The first two figures of each case number indicate the volume number, 33. The third and fourth figures give the number of the week, dating from January 1. The last figure gives the case number for the week. For example, Case 33251 is the first case for the twenty-fifth week of Volume 33.

Abscess of brain, 33261
multiple, 33082
of liver, multiple, 33282
mesenteric, 33332
with perforation and general peritonitis, 33312
myocardial (left ventricle), 33411
subdiaphragmatic and subhepatic, secondary to cholecystectomy, 33101
Actinomycosis of lung, 33052
Adenoacanthoma of cardia of stomach, 33192
Adenocarcinoma, see Carcinoma
Adenoma of bronchus, with hemorrhage into lung, 33322
Adrenal gland, amyloid degeneration of, 33021
Amyloidosis, adrenal gland, 33021
of kidney, 33021
of liver, 33021
of spleen, 33021
of thyroid gland, 33281, 33452
Aneurysm, arteriosclerotic, rupture of, right iliac artery, with retroperitoneal hematoma, 33502
dissecting, with rupture of aorta, 33091, 33121, 33232
mycotic, of brain, 33122
Aorta, see Aneurysm
Appendix, lymphoma of, lymphoblastic, 33292
Arthritis, rheumatoid, 33371
Beriberi disease of heart (probable), 33422
Bile duct, carcinoma of, 33152, 33291
cholangitis of, 33042, 33282
intrahepatic, 33362
defect of, postoperative, 33042
stone in (common), 33282, 33291
Bone marrow, lymphoma of, lymphoblastic type, 33292
Bone, tumor of, Ewing's (rib and toe), 33401
of fibula, 33161
benign giant-cell, of coccyx and sacrum, 33141
Brain, abscess of, 33261
multiple, 33082
aneurysm of, mycotic, with subarachnoid hemorrhage, 33122
embolism of, 33522
glioblastoma multiforme, 33132
infarct of, multiple, 33082
oligodendroglioma (?) of, 33081
(See also Meningitis, Sinusitis)
Bronchus, adenoma of, 33322
papilloma of (basal-cell), with cornification, 33151
Calcification, metastatic (vitamin D intoxication), of kidney and pituitary body, 33071
Carcinoid, multiple, of ileum, with metastases to mesenteric lymph nodes, 33222, 33451
Carcinoma, ampulla of Vater, 33291
of bile duct, 33152
of gall bladder, with perforation, 33341
of jejunum, adeno-, 33201
of kidney, squamous-cell, with nephrolithiasis, 33471
papillary, 33212
of liver (hepatoma), with invasion of cystic duct and metastases to third lumbar vertebra, 33441
metastatic (epidermoid) source undetermined, 33072
of lung, adeno-, metastatic renal-cell, 33012
squamous, 33372, 33432
of lymph nodes, adeno-, metastatic, around bile ducts from previously resected carcinoma of rectum, 33262
of pancreas, 33491
with metastases to liver 33172
with extension into and ulceration of stomach, 33472
of prostate, 33492
of rectum, adeno-, 33432
of sigmoid, with perforation, 33112
of stomach, multiple polypoid, with metastasis to lymph node, 33171
of thyroid gland, giant-cell type, 33512
Cholangitis, see Bile duct
Cholecystitis, see Gall bladder
Choledocholithiasis, see Bile duct
Cholelithiasis, see Gall bladder
Chondroma, of knee, 33041
Chordoma of third lumbar vertebra, 33032
Cirrhosis, see Liver
Colitis, see Intestine
Colon, see Intestine
Cyst, of pancreas, 33321
bronchiogenic, 33461
tracheobronchial, of esophageal wall, 33182
(Diabetes insipidus) 33071
Diverticulitis, see Intestine
Duodenum, see Intestine
Dystrophy, pseudohypertrophic, muscular, 33311
Embolism, cerebral, 33522
mesenteric, 33302
multiple, 33301
pulmonary, 33491
Endocarditis, see Heart
Endometriosis, see Uterus
Esophagus, cyst of, tracheobronchial, 33182
leiomyoma of, 33252
ulcer of, peptic, 33501
Ewing's tumor of fibula, 33161
of rib, 33401
of toe, 33401
Gall bladder, carcinoma of, with perforation, 33341
cholecystitis, chronic, 33291
cholelithiasis, 33291
infarct of, 33241
lymphoma of, lymphoblastic, 33292
Heart, abscess of, myocardial, 33411
Beriberi disease of (?), 33422
Dystrophy of myocardium, pseudohypertrophic, 33311
Endocarditis, bacterial, acute, of mitral valve, 33082
subacute, aortic, 33122
rheumatic, aortic, with stenosis, 33372
mitral, with stenosis, 33131, 33372, 33522
type undetermined, chronic, mitral and tricuspid, 33272
Fibrosis, of myocardium, 33311
diffuse, 33422
Hypertrophy, marked, ventricle, 33131, 33211
Infarct, myocardial, old and recent, 33061
Pericarditis, acute, tuberculous, 33391
Sclerosis, coronary, 33022, 33061, 33231
Stenosis, calcareous, aortic, 33211, 33421, 33511
Thrombus, mural, left auricle, 33522
Hemangioma, cavernous, of lung, 33251
Hematoma, retroperitoneal, 33502
Hematometrium, 33402
Hepatitis, see Liver
Hepatoma, with invasion of cystic duct and metastases to third lumbar vertebra, 33441
Hodgkin's disease, see Lymphoma
Hypernephroma, see Kidney, renal-cell carcinoma of
Ileum, see Intestine
Ileus, gallstone, 33202
Infarct of brain, multiple, 33082
of gall bladder, 33241

of lung, 33301, 33491
of myocardium, old and recent, 33061
Intestine, carcinoid of (ileum), multiple, with metastases to mesenteric lymph nodes, 33222, 33451
carcinoma of, adeno- (jejunum), 33201
(rectum), 33432
colloid (sigmoid), with perforation, 33112
colitis of, ulcerative, 33021
diverticulitis of (sigmoid), acute and chronic, 33062
gallstone ileus, 33202
leiomyoma of (descending colon), 33392
lymphoma of, lymphoblastic (jejunum), 33292
obstruction of (ileum), due to inflammatory adhesions, 33521
ulcer of (duodenum), 33352
volvulus of, 33451
Intoxication, vitamin D, 33071
Jaw, fibrosarcoma of, 33092
Kidney, amyloid degeneration of, 33021
calcification of, metastatic, 33071
carcinoma of (pelvis), papillary, 33212
squamous-cell (with nephrolithiasis), extension into psoas muscle, 33471
necrosis of, diffuse (renal cortex), secondary, 33181
sclerosis, nephro-, 33481
arteriolar, 33231
tumor of, Wilms's, 33242
(See also Nephritis, Nephrolithiasis and Nephrosis)
Knee, chondroma of, 33041
Leiomyoma of colon, descending, 33392
of esophagus, 33252
Liver, abscesses of, multiple, 33282
amyloid degeneration of, 33021
carcinoma of, metastatic (epidermoid) primary source undetermined, 33072
cirrhosis of, alcoholic type, 33331, 33352, 33431
hepatitis, infectious, acute, 33361
hepatoma, with invasion of cystic duct and metastases to third lumbar vertebra, 33441
lymphoma of, lymphoblastic, 33292
Lung, actinomycosis of, 33052
carcinoma of, adeno-, metastatic renal-cell, 33012
squamous-cell, 33372, 33432
embolism of, 33491
embolism of, multiple, 33301
hemangioma of, cavernous, 33251
hemorrhages in, 33322
infarct of, 33301
pneumonia, organizing, 33342
pneumonitis, chronic, 33342
tuberculosis of, chronic, 33051, 33452
miliary, 33071
Lymphoma, malignant, Hodgkin's sarcoma type, of retroperitoneal lymph nodes, 33071
lymphoblastic, of small bowel, appendix, gall bladder, liver, mediastinum, retroperitoneal tissues and bone marrow, 33292
Mediastinum, lymphoma of, lymphoblastic, 33292
Meningioma of spinal cord, 33442
Meningitis, tuberculous, 33071
Mesothelioma of pleura and pericardium, 33111
Muscle, dystrophy of, pseudohypertrophic, 33311
Myocardium, see Heart
Necrosis, diffuse, of renal cortex, secondary, 33181
Nephritis, glomerulo-, acute, 33342
chronic, 33022
subacute, 33482
pyelo-, 33381
Nephrolithiasis, 33471
Nephrosis, hemoglobinuric, 33191
idiopathic, 33372
lipoid, acute, 33101
Oligodendroglioma (?) of brain, 33081
Ovary, oophoritis, and perioophoritis, acute, 33521
chronic, 33162
Pancreas, carcinoma of, 33491
adeno-, with metastases to liver 33172
with extension into and ulceration of stomach, 33472
cyst of, 33321
Pancreatitis, chronic, with extension to wall of stomach, 33102
Papilloma, basal-cell, of bronchus, with cornification, 33151
Periarteritis nodosa, 33181, 33241, 33382
Pericardium, mesothelioma, 33111
pericarditis, acute, tuberculous, 33391
Peritonitis, general, 33312
tuberculous, 33431
Pituitary body, calcification of, metastatic, 33071
Pneumonia, see Lung
Pneumonitis, see Lung
Poliomyelitis, bulbar, 33011
Polyneuritis, porphynuric, 33031
Prostate, carcinoma of, with metastases to spine, 33492
Rectum, see Intestine
Retroperitoneum, neurofibrosarcoma of, 33332
Rib, tumor of, Ewing's, 33401
Sarcoidosis, 33412
Sarcoma, (?) of endometrium, 33221
of jaw (fibro-), 33092
of retroperitoneal tissues (neurofibro-), 33332
of scapular region (fibromyxo-), 33141
Sclerosis, coronary, 33022, 33061
Sinusitis, acute, ethmoid and sphenoid, 33082
Spinal cord, meningioma of, 33442
poliomyelitis, bulbar, 33011
Spine, tumor of (coccyx and sacrum), benign giant-cell, 33141
Spleen, amyloid degeneration of, 33021
hypertrophy of, 33372
Stomach, adenoacanthoma of, 33192
carcinoma of, multiple polypoid, 33171
extension of chronic pancreatitis to wall of, 33102
ulcer, benign, peptic, 33271, 33351
with perforation, 33312
Thyroid gland, amyloid disease of, 33281, 33452
carcinoma of, giant-cell type, 33512
Toe, Ewing's tumor of, 33401
Tuberculosis of lung, chronic, 33051, 33452
miliary, 33071
Tuberculous pericarditis, acute and chronic, 33391
Tuberculous peritonitis, 33431
Tumor, Ewing's, of rib and toe, 33401
of fibula, 33161
giant-cell (see Bone)
Wilms's, of kidney, 33242
Ulcer, of duodenum, with erosion of pancreaticoduodenal artery, 33352
of esophagus, 33501
of stomach, benign, peptic, 33271, 33351
with perforation and mesenteric abscess, 33332
Uterus, cervix, cicatrix of, post radiation, with stenosis of cervical canal, 33462
endometrium, sarcoma of (?) 33221
endometriosis, stromal (endolymphatic fibromyosis Frank) (?), 33221
hematometrium of, 33402
Vein, erosion of (intrahepatic branch of portal), with hemorrhage, 33042
Vertebra, chordoma of, third lumbar, 33032
Vitamin D intoxication, 33071
Volvulus of ileum, 33451
Wilms's tumor, of kidney, 33242

SPEAKERS IN DISCUSSION OTHER THAN TRACY B. MALLORY

The numbers in heavy type refer to cases in which the speaker has presented the Differential Diagnosis.

Adams, F. Dennette, **33052, 33491**
Adams, Ralph, **33151**
Allen, Arthur W., **33042,** 33062, **33222,** 33321
Aub, Joseph C., **33012, 33461**
Ayer, James B., **33011, 33261**
Baker, Myles P., **33482**
Ballantine, H. Thomas, Jr., 33442
Barr, Joseph S., 33041
Bartlett, Marshall K., **33451**
Bauer, Walter, **33091,** 33222, 33311, 33491
Beckman, William W., **33112**
Benedict, Edward B., 33102, 33322, **33501**
Bland, Edward F., **33061,** 33091, **33231,** 33371, 33421
Brailey, Allen G., **33172, 33431**
Burgin, Leo B., **33242**
Burnett, Charles H., **33191**
Burrage, Walter S., 33482
Butler, Allan M., 33081, 33082
Caner, G. Colket, **33132**
Castleman, Benjamin, 33091, 33111, 33131, 33232, 33242, 33262, 33271, 33302, 33322, 33351, 33381, 33382, 33392, 33401, 33402, 33412, 33432, 33451, 33471, 33501, 33512

Chapman, Earle M., **33101, 33251, 33452,** 33511
Churchill, Edward D., **33051**
Clark, Richard J., **33071, 33301, 33481**
Clark, William, **33382, 33441**
Cogan, David G., 33071
Colby, Fletcher H., 33212
Currens, James H., **33122**
Dahl, Lewis K., 33342
Daland, Ernest M., **33161**
Davenport, Lowrey F., **33372**
Dean, Archie L., **33212**
Decker, Briant L., 33502
Dole, Vincent P., **33352**
Donaldson, Gordon A., **33312**
DuToit, Charles H., 33381
Ellis, Daniel S., 33172, **33262,** 33321, **33362,** 33431
Evelyn, Kenneth A., 33231
Faxon, Henry H., 33301
Franseen, Clifford C., **33401**
Giddings, W. Philip, 33152, **33302**
Goldman, Henry M., **33092**
Hamlin, Edward, Jr., **33202, 33332, 33512**
Ingersoll, Francis M., **33402,** 33451
Jacobson, Bernard M., **33102, 33201, 33331**
Jeffries, William McK., **33412,** 33512
Jones, Chester M., **33072, 33252**
Kalil, Toufic H., 33142, 33241, 33291, 33301, 33442, 33491, **33492**
King, Donald S., 33012, **33111,** 33151
Kranes, Alfred, **33281, 33321,** 33382
Kubik, Charles S., 33011, 33031, **33032,** 33071, 33081, 33132, 33261, 33382, 33442, 33492
Leech, Clifton B., 33091
Lingley, James R., 33032, 33092
Linton, Robert R., 33282, **33291**
Ludwig, Alfred O., **33371**
McGinn, Sylvester, **33211**
McKittrick, John B., 33112, 33172, **33192**
McKittrick, Leland S., **33152**
Meigs, Joe V., **33462**
Michelsen, Jost J., **33081,** 33261
Miller, Carroll C., 33151, **33322**
Mixter, C. J., Jr., 33351
Moore, Francis D., **33062, 33472,** 33491
Palmer, Robert S., 33231
Paul, Oglesby, **33232, 33522**
Pickering, George W., **33391**
Pittman, Helen S., 33291, 33461
Reyersbach, Gertrud C., 33272, **33422**
Richardson, Wyman, **33082, 33241, 33361,** 33362
Risley, Thomas S., 33202
Robbins, Laurence L., 33012, 33052, 33222, **33392,** 33501
Robertson, Charles W., 33171, 33281
Ropes, Marian W., **33021,** 33102, **33311,** 33371
Rose, Augustus S., **33031,** 33132, **33492**
Ross, Ralph A., **33272**
Sarris, S. Peter, **33341**
Scannell, John G., **33342**
Schall, Leroy A., **33182**
Schatzki, Richard, 33042, 33071, 33101, 33102, 33111, 33131, 33151, 33152, 33171, 33271, 33321, **33351**
Schier, W. Wilson, **33432**
Schulz, Milford D., 33132, 33331, 33342
Short, Charles L., **33022, 33181, 33411**
Simeone, Fiorindo A., **33471, 33502**
Simmons, Channing C., **33141**
Simmons, Fred A., **33162**
Smith, George G., 33191, 33502
Sniffen, Ronald C., 33251
Sosman, Merrill C., 33032, **33041,** 33092
Soutter, Lamar, 33372
Sprague, Howard B., 33121, 33131, **33511**
Stillman, J. Sidney, **33282**
Sweet, Richard H., 33051, 33182, 33251, 33252, 33501
Sweet, William H., 33492
Taylor, Grantley W., 33041, **33142,** 33241
Taylor, Isaac M., 33031, 33311
Thoma, Kurt H., 33092
Towery, Beverly T., 33052
Townsend, James H., **33292**
Ulfelder, Howard, 33402
Volwiler, Wade, 33042, 33072, **33271,** 33331, 33431
Watkins, Arthur L., **33442**
Welch, Claude E., **33171, 33521**
White, James C., 33261
White, Paul D., 33121, **33131,** 33272, 33391
Williams, Conger, **33121,** 33391, 33411, **33421**
Wilson, Norman J,, 33142
Wyman, Stanley M., 33181, 33191, 33192, 33201, 33202, 33221, 33251, 33252, 33261, 33311, 33312, 33332, 33341, 33361, 33362, 33371, 33372, 33381, 33401, 33411, 33412, 33432, 33452, 33461, 33471, 33481, 33482, 33502, 33521, 33522
Younge, Paul A., **33212**
Zeller, J. Wallace, **33381**

NOTICES (*Continued from page 1004*)

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Thursday, January 1

Friday, January 2
*10:00 a.m.–12:00 m. Medical Staff Rounds. Peter Bent Brigham Hospital.

Monday, January 5
*12:15–1:15 p.m. Clinicopathological Conference. Peter Bent Brigham Hospital.

Tuesday, January 6
12:00 m. X-Ray Conference. Margaret Jewett Hall, Mt. Auburn Hospital, Cambridge.
*12:15–1:15 p.m. Clinicoroentgenological Conference. Peter Bent Brigham Hospital.

Wednesday, January 7
*9:00–10:00 a.m. Histochemical Studies of Blood and Bone Marrow. Dr. Jack Rheingold. Joseph H. Pratt Diagnostic Hospital.
*12:00 m. Grand Rounds and Clinicopathological Conference (Children's Hospital). Amphitheater, Peter Bent Brigham Hospital.
*2:00–3:00 p.m. Combined Clinic by the Medical, Surgical and Orthopedic Services. Amphitheater, Children's Hospital.
8:00 p.m. Tufts Alpha Omega Alpha. Boston Medical Library, 8 Fenway.

*Open to the medical profession.

January–April. Thirteenth Postgraduate Seminar in Neurology and Psychiatry. Metropolitan State Hospital. Page 348, issue of August 28.

January 7. Tufts Alpha Omega Alpha. Page 1004.

January 7–30. Joseph H. Pratt Diagnostic Hospital. Medical Conference Program. Page 1004.

January 8. New England Hospital for Women and Children. Page 1004.

January 8. Dysmenorrhea. Dr. Joe V. Meigs. Pentucket Association of Physicians. 8:30 p.m. Haverhill.

January 14. Phi Delta Epsilon Lecture. Page 968, issue of December 18.

January 20 and 21. American College of Surgeons. Commodore Perry Hotel, Toledo, Ohio. Page 930, issue of December 11.

January 26 and 27. American College of Surgeons. Ansley Hotel, Atlanta, Georgia. Page 930, issue of December 11.

January 30 and 31. American College of Surgeons. Oklahoma Biltmore Hotel, Oklahoma City. Page 930, issue of December 11.

January 30 and 31. Conference on Normal and Pathologic Physiology of Pregnancy. Page 1004.

February 6. American Board of Obstetrics and Gynecology. Page 242, issue of August 14.

March 28–April 4. American Association of Industrial Physicians and Surgeons, American Industrial Hygiene Association, American Conference of Governmental Industrial Hygienists, American Association of Industrial Nurses, Inc., and American Association of Industrial Dentists. Hotel Statler, Boston.

April 19–23. American College of Physicians. Page xiii, issue of July 31.

May 6–8. American Association for the Study of Goiter. Page xiii, issue of July 31.

May 17–20. American Urological Association. Hotel Statler, Boston.

May 18–22. American Association on Mental Deficiency. Copley Plaza, Boston.

May 25–27. Massachusetts Medical Society. Annual Meeting. Hotel Statler, Boston.

District Medical Societies

Franklin

January 13.
March 9.
May 11. Annual Meeting. Hotel Weldon.
All other meetings will be held at Franklin County Hospital.

Middlesex East

January 21.
March 24.
May 12. Annual Meeting.
All meetings will be held at the Bear Hill Golf Club.

Norfolk

January 27. Round-Table Discussion: Bleeding from the alimentary tract.
February 24. Obstetric and Gynecologic Night.
March 23. Harvard Night.

Plymouth

January 15. Brockton Hospital, Brockton.
February 19. Toll House, Whitman.
March 18. Goddard Hospital, Brockton.
April 15. State Farm, Bridgewater.
May 20. Lakeville Sanatorium, Lakeville.

Worcester

January 14. St. Vincent's Hospital.
February 11. Worcester State Hospital.
March 10. Memorial Hospital.
April 14. Hahnemann Hospital.
May 12. Annual Meeting.

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